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BRONCHOSCOPY IN PULMONARY TUBERCULOSIS.

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Tuberculosis of the trachea and main bronchi as a clinical subject has only comparatively recently appeared in the literature. For a long time there were only reports of autopsy material. Since the introduction and common use of bronchoscopy it has been recognized during life. This explains the varying statements as to its frequency. Myerson, in 1928, reporting a case of tuberculous ulceration of the bronchus in lung abscess following tonsillectomy, stated that the condition was rare. Bugher, writing in the *American Journal of Medical Sciences* in April of this year, stated that in 122 cases of pulmonary tuberculosis studied at autopsy, 41 per cent showed lesions of the trachea, main bronchi, or both. As bronchoscopic examination becomes more frequent, the condition is found more and more frequently during life. Epstein and Ornstein, reporting on the bronchoscopic examination of 150 cases of tuberculosis at the Sea View Hospital, wrote a very comprehensive paper on this subject in 1936. They pointed out that very little emphasis had been placed on tuberculosis of the trachea and main bronchi as a complication of chronic pulmonary tuberculosis and that the reason for this was no doubt failure to recognize the condition during life.

As to pathogenesis, ideas vary somewhat. Reichle and Frost consider that the most frequent mode of origin is by

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contiguity. The lymphatics surrounding the major bronchi and trachea and the mediastinum absorb infectious material in the periphery of the lung. In the mediastinum they are in close contact with the mucous glands lying in the walls of the trachea and bronchi outside the cartilages. Owing to this close contact of glands and diseased lymphatics, the glands are frequently attacked and carry infectious material through the walls of the bronchi into the mucous membrane. By continuity these authors mean the extension of tuberculosis along the mucous membrane of large bronchi leading out of tuberculous cavities. The most infrequent mode of infection they consider to be by implantation from the sputum. In their opinion, the mucous membrane with its cilia, aided by the cough, opposes a stout barrier against infection. Bugher, on the other hand, in his article states that implantation from the sputum is the most frequent way of infection.

The clinical classification is made according to the site of the first attack. If the infection is by implantation, and this undoubtedly occurs, the disease appears as large or small, deep or shallow ulcers in the mucous membrane. The question can be raised as to whether or not the use of pneumothorax and other methods of treatment designed to put the lung at rest do not promote the appearance of ulcers, for it must be remembered that a lung at rest no longer has at its command a powerful cough to clear the bronchi of sputum. It may easily come about that sputum depending for its removal on the cilia only will lie much longer in contact with the mucous membrane than if the cough were also present, hence there is increased opportunity for invasion of the mucous membrane and the production of ulcers.

The ulcers have a natural tendency to heal. In healing they produce scar tissue and so arises the second class of case, that with shrinking and formation of scar tissue in the bronchi. These cases when bronchoscoped may show a bronchus narrowed or completely closed by a dense mass of scar tissue. The clinical appearance of the case will depend on the presence or absence of cavities beyond the stricture and on whether or not those cavities, if present, are infected. If infected, they will show all the symptoms of an undrained lung abscess, with cough, fever, occasional expulsion of large quantities of pus, if the bronchus is more or less open, and frequently severe pain in the chest.

Stricture of the bronchus may be brought about in another way. If the mucous membrane is infected by continuity, the disease creeps along under the surface without breaking through and forms ulcers. The consequent great increase of thickness of the mucous membrane will almost close the lumen of the bronchus and bring about the symptoms of poor drainage. If this process heals, shrinking and contraction will cause a great distortion of the bronchial tree.

In speaking of pathogenesis, the existence of peribronchial infection was mentioned as bringing about infection by contiguity owing to the presence of diseased lymphatics and lymph nodes about the walls of the main bronchi. A defensive proliferation in the walls of the bronchi might close this avenue of infection, but contraction of the defensive tissue would in turn distort the bronchi and cause obstruction of the lumen.

Another clinical appearance is caused by the presence of diseased and enlarged lymph nodes in the mediastinum. These may increase to such a size as to close a main bronchus by pressure. At times these glands will break down and erode their way into the lumen of the bronchus. Then appear all the symptoms of an obstructed bronchus, with collapse of the lung supplied by that bronchus, and if a sufficiently large amount of lung is collapsed, the clinical symptoms of severe dyspnea and cyanosis. Should the disease of the bronchial wall be sufficiently severe, the supporting cartilages may be destroyed and then the shrinking and distortion are of extreme degree.

Another form which tuberculosis of the bronchi may take is the formation of tumors in the bronchi. These may be on the bed of an ulcer or take on the appearance of a tumor springing out of an intact mucous membrane. The symptoms caused by a tumor will be according to its size. A small tumor may manifest itself only by hemorrhage. A pedunculated tumor may give a bizarre symptom of a fluttering noise at the mouth on deep breathing or coughing. If the tumor is obstructive, but not completely so, wheezing may be complained of. If a large bronchus or the trachea is obstructed, the complaint may be of dyspnea and cyanosis so closely resembling asthma that the diagnosis of asthma is frequently made.

To sum up, then, the ways in which tuberculosis may attack the trachea and main bronchi: There may be shallow ulcerations; tuberculous tumors within the lumen; a general thickening and distortion of the wall of the trachea or bronchi; and finally pressure from the outside by enlarged lymphatic glands. The healing of the ulcers may cause such formation of scar tissue as to close the lumen.

In discussing diagnosis, we will first consider the securing of sputum. There will frequently be cases, especially children, in which the existence of tuberculosis will be suspected and yet it will be impossible to confirm that diagnosis by finding tubercle bacilli in the sputum. Tuberculosis frequently narrows or completely closes the main bronchi. This comes about in three ways: First, through pressure from the outside by enlarged lymph nodes; second, by a filling of the bronchus by tuberculous tumors; third, by the formation of scar tissue when the tumors or ulcerations heal.

In such cases the sputum ordinarily coughed up is that coming from the trachea and the other bronchus. This sputum will show only the ordinary mixture of pyogenic organisms. On the other hand, the secretion beyond the closure of the bronchus may be loaded with tubercle bacilli. In solving such a problem the bronchoscope is useful. Passing the tube down to the stricture, one will at times find it possible to pass it beyond the stricture, pushing aside the soft swollen glands. Aspiration, then, of the pus from beyond the stricture will secure a positive sputum.

Let me quote here a case in illustration of such a condition. This baby, age 10 months, was admitted to the Babies' Hospital on April 8, 1927, with complaints of cough and fever of one month's duration. Before admission the left chest had been aspirated and pus was said to have been obtained. The signs over the chest suggested a pyopneumothorax. X-ray of the chest showed consolidation of the left upper lobe and the appearance of thickened pleura. The right lung showed a mottling, which made one suspect small bronchopneumonic areas, possibly of tuberculous origin. Smears of what sputum could be obtained showed no tubercle bacilli. While the child was in the hospital, the chest was aspirated several times, and no pus was obtained. The tuberculin reaction was positive. Two weeks after admission the

child was bronchoscoped. The trachea was found displaced to the right; a small amount of white pus was traced to the left main bronchus; the orifice of the left main bronchus was narrowed to a slit by outside pressure from the left and above. The narrow part was passed with the tube and then the lumen was found to be normal in size. The mucous membrane was intensely congested. There was a small amount of pus in the bronchus, of which a specimen was taken. The orifice of the left upper lobe bronchus looked normal. The tentative diagnosis made was enlarged lymph nodes of tuberculous origin. Smears of the pus secured at this bronchoscopy contained tubercle bacilli. That is the important thing to note in connection with this case. Smears of the sputum coughed up had been negative for tubercle bacilli; smears of the sputum secured during bronchoscopy from beyond the narrowed bronchus were positive for tubercle bacilli.

At times positive smears may be secured through bronchoscopy when no visible sputum is raised. Allow me to quote a case to illustrate this point. A young woman, age 20 years, had had during the course of a year a number of obscure illnesses. Three months before seen by this author, she had had an attack of what was thought to be appendicitis, accompanied by pain in the region of the appendix and loss of appetite. During the course of a six weeks' illness, the pain shifted to the right lower thorax. She was thought to have pleurisy or pneumonia. Her chest was tapped several times and a small amount of fluid removed. A dry cough was present; never any expectoration. At times she developed a temporary collapse of the right lung, suggesting a plugging of a bronchus. With the thought in mind of nonopaque foreign body, we bronchoscoped her. The mucous membrane of the mouth of the right upper lobe bronchus was found to be intensely red, swollen, with a suggestion of ulceration about it. A smear was taken of this area. This smear contained tubercle bacilli.

I am not suggesting that the diagnosis in these related cases could not have been made without the use of the bronchoscopy—I merely wish to point out that the bronchoscopy made the diagnosis at once, whereas it had been uncertain for weeks in the one case and for two or three months in the other.

Now, as to the blocking of the bronchi by tuberculous tumors, all lung diseases give practically the same symptoms. That is to say, tuberculosis, benign or malignant tumor, retained foreign body, or what you will, are likely to give cough, pain in the chest, shortness of breath, excessive expectoration. Physical signs are much the same in all. Even the X-ray appearances are not characteristic. All tumors of the bronchi, if large enough to obstruct the bronchus, give symptoms of obstruction of the bronchus, such as shortness of breath, cough, on physical examination the signs of massive collapse and on X-ray examination the appearances that go with this latter condition. If there is retained secretion and infection, symptoms of sepsis will be present. At times a bronchoscopy will make the diagnosis promptly, either by securing positive sputum from beyond the tumor or by securing a biopsy of the tumor.

A woman, age 44 years, came to Dr. James A. Miller in November, 1934, with the following story: Eight years before, she had had a thyroidectomy (note that). Five months before she saw Dr. Miller she had noticed a wheezing sensation in her throat and shortness of breath, what she called an asthmatic condition. She had had no chest symptoms and was not subject to colds. Having had a thyroidectomy and now having difficulty that seemed to be connected with her throat, she naturally thought that something related to the thyroid gland or the thyroid operation had arisen to give her her symptoms—some late complications. Her first impulse was to go to the doctor who had operated on her thyroid gland. He, finding nothing wrong in connection with the operation, had sent her to a laryngologist. He treated her for some time and then, the symptoms becoming worse, sent her to Dr. Miller for examination of her lungs. His X-ray films showed a lesion of the right upper lobe and the pulling of the mediastinum toward the right side, which suggested a plugging of the right bronchus and collapse of the right upper lobe (note also the widening of the mediastinum). Remember now, this woman had had no chest symptoms and no sputum. Her symptoms were laryngeal. Tuberculosis had not been suspected. She was bronchoscoped. An intense congestion of the tracheal mucous membrane was found and distortion of the bifurcation by compression from the outside. There was also considerable fixation of the tracheobronchial tree, sug-

gesting neoplastic infiltration. A very vascular mass of neoplastic tissue was found plugging the orifice of the right upper lobe bronchus. A biopsy was taken of this material. The following note was made: "In the interpretation of these appearances one would say that there was great infiltration of the mediastinal structures, between the main bronchi and about the right main bronchus. This might be either inflammatory or neoplastic. This infiltration has closed by compression the right upper lobe bronchus. The diagnosis will be made on the biopsy. If it turns out to be carcinoma, the question is settled; if it turns out to be granulation tissue, all the appearances could be accounted for by supposing that there is a lung abscess in the right upper lobe. This is the less likely of the two diagnoses." You will see that the diagnosis was not made positively, even on the bronchoscopy. Lung abscess and carcinoma were most thought of. The tissue proved to be tuberculous. This woman is now under treatment for tuberculosis and is getting on very well.

Another case of tuberculous tumor causing obstruction which I should like to quote is that of a young woman, age 24 years, who came to Presbyterian Hospital in October, 1931. She had a family history of tuberculosis. She had been admitted twice before to the Presbyterian Hospital for rather indefinite illnesses. Tuberculosis had not been suspected. For about five months prior to this admission she had had a dry, hacking cough without sputum. The physical examination of the lungs was practically negative, according to note made Aug. 6 by Dr. Lamb. She had no fever. X-ray films of the chest showed the left upper lobe to be a little emphysematous. On fluoroscopy the mediastinum moved a little to the left with inspiration. As she breathed, a fluttering rale and a wheeze could be heard with the stethoscope placed at her mouth. A hand on the chest could feel a respiratory vibration. These were the signs of the partial obstruction of the left upper lobe bronchus, possibly by a nonopaque foreign body. Bronchoscopy showed an ulceration on the left wall of the trachea and the left main bronchus down to the left upper lobe bronchus. There was a loose membrane on it, which flapped to and fro in the wind of her breathing like a pedunculated tumor. Smears and cultures were taken from the ulcerated area, which showed tubercle bacilli. This patient went to Trudeau for treatment and in July, 1934, had almost completely recovered.

I said before that pulmonary diseases all have symptoms in common. The previous case quoted had the symptoms of carcinoma of the lung, and it was thought to be that, even on bronchoscopy. The case just quoted had the symptoms of nonopaque foreign body.

Now, I go on to describe those cases in which the obstruction of the bronchus is caused by the scar tissue of healed ulcers.

A patient was referred by Dr. Miller in September, 1935. She had been under treatment for tuberculosis of the left lung for some time and had had pneumothorax done. She was running a septic type of temperature. Bronchoscopy showed the left main bronchus to be almost completely closed by scar tissue. Plugging the narrow lumen was a mass of granulation tissue. This was removed with forceps and proved to be tuberculosis. A suction tube was then introduced into the stricture and a large amount of pus sucked away. This pus contained tubercle bacilli. The patient's response to this treatment was very favorable. She ceased to have a septic type of temperature; cough and expectoration stopped. The question now arose as to what was best to do. Here was a patient with a strictured bronchus, with an abscess of the lung or a bronchiectasis beyond it. It was finally decided that an attempt should be made to enlarge the lumen by stretching it, at the same time controlling the suppuration by aspiration. This has been carried out. Rather frequent bronchoscopies have been done and dilation of the stricture attempted. The result as far as enlarging the lumen is concerned has been failure. The lumen continues to be small and the scar tissue so tough that it cannot be stretched. The suppuration, however, has been controlled by the repeated aspirations. The patient is free from cough, fever and expectoration. The thing we may expect in connection with healing of tuberculous ulcers then is closure of the bronchus and the development of suppuration beyond with all the symptoms of pulmonary abscess. The treatment I suggest is stretching of the stricture and aspiration of the pus.

This case just quoted illustrates the result of spontaneous healing of a tuberculous ulcer. I shall now describe a case which healed under treatment and contracted. This patient

came to Dr. Miller in August, 1935. Three weeks prior to that time she had had a gastric upset. At that same time she had X-rays taken of her chest. She was told that there were suspicious shadows suggesting an inactive tuberculosis. More recently she had begun to feel tired and to have a dry cough. She was bronchoscoped in February, 1936. The bronchoscopy disclosed a great ulceration of the left main bronchus, a tumor formation, which closed the bronchus; a biopsy was taken. There was also a swelling of the larynx, which suggested tuberculosis of the larynx. Note that this patient was bronchoscoped in spite of the suspicion of tuberculosis of the larynx. The bronchoscopy was repeated about a week later. It was noted that the ulceration showed less heaping up of granulation tissue than at the last bronchoscopy. This might have been the effect of the silver nitrate. I have had it in mind that perhaps the application of silver nitrate would benefit these cases. It appeared to help this case and would be milder treatment than diathermy. Silver nitrate, of course, does not help any other tuberculous granulations. It certainly would be a less severe treatment than diathermy, which is not without its dangers, as I shall show later. The bronchoscopy was repeated after two weeks. It was found that the mucous membrane was much less inflamed than at the former bronchoscopy; the lumen was wide open. One or two areas of granulation tissue were coagulated with the diathermy current. This patient was then bronchoscoped two months later. The ulceration was found to be completely healed over with smooth, firm scar tissue. The lumen was reduced to about a quarter of an inch in diameter. A suction tube was introduced through this narrow lumen and no blood or pus came from beyond it. The present condition is a healed, contracted bronchus and a dry lung beyond. This case makes one think that perhaps too vigorous coagulation of tuberculosis ulceration in the bronchus might promote the formation of scar tissue and the contraction of the bronchus. However, it should be noted that this same contraction of the bronchus had occurred in a case which had healed spontaneously.

I shall now describe a more successful case of treatment of tuberculous ulcers of the bronchus. This woman, age 34 years, was admitted in July, 1935, to Lenox Hill Hospital.

She had been under treatment for a number of years for tuberculosis of the left lung. She had had no symptoms for almost four years. In the past few months, however, she had developed a cough and a wheeze (note the frequency of wheeze, one of the symptoms of foreign body). She was raising a small amount of sputum, which showed some tubercle bacilli after having long been negative. Examination of her chest showed the right lung to be clear and the left lung completely collapsed. On account of the wheeze, it was thought there might be some obstruction in the larynx secondary to a tracheotomy which she had had done in childhood. The bronchoscopy showed one inch down the left main bronchus on the outer wall a shallow ulceration, and at the lower end of this a mass of soft vascular tissue obstructing the lumen of the bronchus. This mass bled easily, but not profusely on contact with the tube. A biopsy was taken from this examination. The tissue proved to be tuberculous. This patient had three subsequent bronchoscopies. One on July 16, 1935, when the ulcerated area was coagulated by diathermy; one two weeks later, when the previous treatment was repeated; one six months later, when it was found that the ulcer had healed and the tumor mass had disappeared, leaving a wide open lumen. The patient was feeling very well.

Now, the fact that a case healed spontaneously brings up this thought: If these cases will heal themselves, why submit them to bronchoscopy, once the diagnosis is made? I am willing to leave that question open to discussion; though in favor of bronchoscoping them, I call your attention to the fact that those that have been bronchoscoped and treated have improved very rapidly.

Yet, there are dangers in the treatment by bronchoscopy and coagulation. I have already pointed out that severe stricturing may occur. There is also the danger from hemorrhage. I had under treatment a young woman with a tuberculous tumor of the right bronchus. She was treated several times by bronchoscopy and coagulation, and her ulcer healed rapidly. At the last bronchoscopy one small granulation was coagulated. A moderately profuse hemorrhage resulted in her death. At autopsy it was found that the smalled bronchi were plugged by blood clots. Her cough failed to expel the blood and aspiration was not successful.

As to treatment, as proved by autopsy, tuberculous lesions of the trachea and bronchi will heal spontaneously. Moreover, after the diagnosis has been made by bronchoscopy, it has come about that the symptoms will disappear and the patient spontaneously recover. As bronchoscopy is more frequently done in tuberculous cases, it is found that in many cases areas of scar tissue show that tuberculous ulcers have some time been present; hence arises the question of treatment. Some authors even say that no treatment other than that of tuberculosis in general is indicated or needed, especially in those cases where there are no severe symptoms. Suppose, for instance, that in a case with positive sputum, doing fairly well, a small ulcer is found in the bronchus: Is one in such a case justified in repeating the bronchoscopy in an endeavor to heal the ulcer, which may, after all, heal spontaneously? The answer of this author is yes. The ulcer may, indeed, heal spontaneously, but, on the other hand, it may not. It may increase in size and then heal spontaneously. Under these conditions the ulcer contracture might put the other lung out of business, as well as the diseased lung, so it would appear that the thesis that small ulcers should be treated can be defended.

Several methods of treatment have been used by this author for flat, shallow ulcers. That first used was coagulation with diathermy. This was found to work well in a number of cases, but there are two objections to it. The ulcer scar tissue may be excessive and it may be dangerous.

The removal of tumors which are obstructing the bronchi needs no defense. All will agree that such lesions should be removed. They may be removed either by biting forceps, applications of silver nitrate or coagulation. In cases where the obstruction of a bronchus leads to damming up of secretion and the formation of secondary abscesses beyond, the indication for bronchoscopy and aspiration is thoroughly recognized. In such cases, repeated bronchoscopies with stretching of the stricture and aspiration are justified.

I wish to present here a suggestion for dilating these strictures. It is known that the application of the negative pole of a galvanic current will soften scar tissue. I have had made an applicator for passing through the bronchoscope with different size copper tips. With this applicator succes-

sive sizes can be applied to a stricture for about three minutes at a session, with the possibility of permanently enlarging the lumen. I have used this method as yet in very few cases. I know that the use of the ordinary glove stretcher dilator and ordinary sounds is unsuccessful, as these strictures invariably contract again after stretching.

Where the obstructing agent is a mass of thick mucus or mass of cheesy material from tuberculous cavities which have been forced into the main bronchus, removal by aspiration hardly needs any defense. In a similar way, obstruction of a bronchus by rupture into it of a gland should be relieved by aspiration through a bronchoscope.

The treatment of the shallow ulcers by coagulation has already been mentioned. Application of silver nitrate, 10 per cent, to these ulcers has already been used as a method of cure. It has been shown, however, that the application of silver nitrate to bronchi may result in the formation of scar tissue and even strictures may be reduced. The aim of the treatment should be the healing of the ulcer with as little scar formation as possible.

I wish to suggest here another method; that is, the application of a quartz rod through the bronchoscope. With an applicator which has a small quartz bulb at the end, quartz rod rays may be applied to the surface of the ulcer. The use of such light in the larynx is well known and proven to be of benefit. There is no therapeutic reason why it should not also benefit tuberculous ulcerations in the trachea and bronchi. I have used this method on only a few cases and over a short period, so that it has hardly been proven, yet it appears to be worth while.

Summing up, then, we have the treatment of ulcers by coagulation, silver nitrate and the quartz rod; the treatment of strictures by stretching with copper ionization; the treatment of tubercular tumors by removal with forceps or coagulation; the treatment of obstruction, caused either by a mass of mucus or a mass of cheesy material in cavities, or by a mass of cheesy material in ruptured glands, by aspiration; the treatment of secondary abscesses or of bronchiectasis beyond strictures by aspiration.

TUBERCULOSIS OF TRACHEA AND BRONCHI.

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CAUSED BY EROSION FROM LYMPH NODES.

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THE COMMON COLD, ETIOLOGICAL FACTORS AND THEIR RELATIONSHIP TO OTHER RESPIRATORY TRACT CONDITIONS.

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Generally the common cold as such is evident first in the upper respiratory tract, with its well-known train of local and general signs and symptoms.

Repeated common colds as usually understood practically are limited to human beings. Thus far, search for an animal in the lower scale that is susceptible to the repeated common cold has not been successful.

We may well ask ourselves the reason for this obvious fact, and an analysis of possible differences between the upper respiratory tracts of human beings and various animals may perhaps aid us in the solution of this problem. The analysis should give, first, precipitating physical factors; second, bacteriological and immunological factors; and third, anatomical differences in the upper respiratory tract structures between the human and other animals. These may be grouped as follows:

ETIOLOGICAL FACTORS IN COLDS.

normal or immune barrier

Physical	Pathologicoanatomical	Infection
Precipitating factors affecting other two factors, either locally or by involving breakdown in general immunity.	(Underlying key factor) Abnormal turbinate-septal - mural structures (contact). Infective lymphoid tissues in nose and pharynx.	Bacterial and virus-symptom producers.

The first and second etiological factors have been voluminously considered in the Annals of the Pickett-Thomson Research Laboratory[1] up to 1932, as well as in other articles up to the present time, and may be briefly summarized as follows:

1. Physical factors, such as exposure, etc., may be precipitating ones affecting the other two factors, either locally

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or by involving a breakdown in general immunity. They do not differ for humans or domesticated animals.

2. a. The infection factor—no definite universal organism or virus has been decided upon as the sole cause of common colds limited to man. The bacteria or virus are probably the symptom producers locally and generally.

b. As to species immunity, it is possible that the type or method of immunologic response in the human being or some other factor is not present or has not been uncovered in any of the lower animals. A decision on this particular question necessarily awaits the results of further investigation.

3. Differences in structure and function of the upper respiratory tract of human beings and other animals: What relationship if any do the pathologicoanatomical structures of the upper respiratory tract have to the common cold? Gafafer[2] states: "The group with tonsils and adenoids, and the group without tonsils and adenoids, presented no significant difference with respect to *1.* frequency, *2.* severity, *3.* type of attack of disease of the upper respiratory tract (common cold)."

My observations lead me to believe that the pathologico-anatomical factors have a very important bearing upon the common cold.

Studies of the comparative anatomy of the respiratory tracts of domesticated animals and human beings reveal pronounced differences. In none of the domesticated animals are the external structures of the nose as vulnerable and exposed to trauma as are those of the human being. The bone and cartilage in infancy and early childhood are very thin. The cartilage is pliable and the osseous structure is very friable. At this stage of life the human is comparatively helpless in protecting himself against injury to these structures; consequently, the internal as well as the external nasal structures of the young child are subject to frequent injury and disarrangement. A slight blow on the nose may result in fracture of the nasal bones or bending of the very thin septal cartilage, and subsequent interference with respiratory function.

In infancy and early childhood, the nasal passages are small in relation to the size of the body. In the immature and unstable infant, with narrow nasal passages, unstable responses to thermostatic effects on the internal nasal tissues ("air conditioners") are manifested by a relatively greater rhinorrhea and sneezing, due to contact between the swelling turbinates and the nasal septum. This is the manner in which the human being controls the intake of air. In the lower animals, in addition to the turbinates, the contractile nostrils function also as "air conditioners."[3] In these also, it is almost universally true that the external nasal structure affords markedly better protection against trauma than does that of the human. The septum is appreciably thicker and better protected against injury because of its inherent make-up, as well as its placement in relation to other bones and intranasal tissues. Chauveau[4] shows that the turbinate structures are much better protected by their anatomical position, both in relation to the exterior of the nose and the cartilaginous septum, and are usually a part of the nasal accessory sinuses. The anatomy and histology seem to differ in order to take care of the different physiological needs. Smell in the human being is relatively unimportant and respiratory regulation very important. The human requires the upper respiratory tract chiefly for breathing and not for the same degree of olfactory needs as the animal, which may explain the high development of this portion of the upper respiratory tract in lower animals.

In most of the domesticated animals, studies of the lymphatic and vascular circulations of the upper respiratory tract reveal fine superficial lymphatics (the existence of which were for a long time doubted), which finally drain into the submaxillary glands from the turbinate and septal structures. Large anastomosing veins are found in the deep layer of the mucosa, forming a long close and magnificent plexus, terminating in the satellite vein of the nasal artery, marked at certain points as at the appendages of the turbinate bones, giving the mucous membrane somewhat the appearance of erectile tissue. This favors the stagnation of blood. Microscopically, the tunica propria of the Schneiderian membrane (regio respiratoria) is seen to contain many vessels and glands of the racemose or mucous type over the turbinates and septum, though these are rare or absent on the external

face of the turbinates, where ciliated and stratified epithelium cover this. The olfactory mucous membrane (regio olfactoria) is thinner and more delicate. Its tunica propria contains tubular glands (glands of Bowman), and the epithelium is columnar, stratified and devoid of cilia.

In the human being, the most vulnerable parts of the upper respiratory tract are in the various portions of the thin nasal cartilage of the septum, especially where this cartilage joins the bone or other structures along the floor of the nose, the nasal vestibule, at the vomer and ethmoid junctions, as well as near the nasal bones. Aside from deviation of the cartilage itself due to abnormal crowding of the compressed cartilage, with or without fracture of this structure, the most common lesions are septal spurs or ledges near the junction points previously mentioned, encroaching upon the nasal areas to such a degree that slight thermostatic action, with resultant enlargement of the turbinates, causes immediate turbinate-septal-mural contact, with a subsequent excessive mucus formation etc. Except in a small percentage of cases, such anatomical abnormalities are limited to the area between the front portion of the nose to within one-quarter to one-half inch past the anterior edge of the middle turbinates.

With regard to the human anatomy of the upper respiratory tract, Piersol[5] states: "A rich lymphatic network lies beneath the mucous membrane of the nasal cavities and from it vessels pass in two directions. Those of the anterior and lower portions of the fossae pass forward, and partly at the external nares and partly by passing between the nasal bones and the cartilages, communicate with the superficial nasal lymphatics. The majority of the vessels, however, take a backward course, terminating in different node groups. Some join the vessels draining the palate and tonsils to pass to the superior deep cervical nodes and especially to that one which is situated in the angle formed by the union of the facial and internal jugular veins, while the rest unite to form two to four stems which pass over the lateral surface of the pharynx and terminate in the retropharyngeal nodes. The lymphatics of the sinuses which open into the nasal cavities follow, in part at least, the same course as those of the nasal mucous membrane, their principal termination being in the larger retropharyngeal nodes." "All these deep cervical nodes receive

the efferent vessels from the superficial nodes (and thus from their tributaries mentioned above), as well as other lymphatics of the head and neck, retropharyngeal, suprahyoid, etc."[6] "Stems emerging from the network (of the soft palate) pass toward both surfaces of the palate, those lying below the upper respiratory surface passing backward and outward to join the stems from the nasal mucous membrane just below the orifice of the Eustachian tube, whence their course is similar to that of the nasal stems. Some of them pass upward and backward to perforate the superior constrictor of the pharynx and terminate in the lateral retropharyngeal nodes, while others descend beneath the mucous membrane covering the posterior pillars of the fauces and after perforating the superior constrictor terminate in the upper nodes of the superior deep cervical group."[7] "Those of the pharynx are also abundant, especially above. The stems which arise from the roof and upper part pass principally to the retropharyngeal nodes."[8] "The retropharyngeal nodes are for the most part small, appearing as slight enlargements of the lymphatics which drain the posterior surface of the pharynx. In addition to these 'intercalated nodes,' however, one or two much larger nodes occur at the junction of the lateral and posterior surfaces of the pharynx."[9] Edington[10] makes these statements: "The mucoperiosteum anteriorly drains externally to the submaxillary glands and posteriorly to the superior deep cervical glands and the lateral retropharyngeal glands. . . The nasal lymphatic drainage is to superior deep cervical lymph glands, and some to the retropharyngeal lymph glands, hence arise retropharyngeal abscesses."

With regard to lymphatic structures, Yates[11] states that there is a small quantity of this tissue beneath the epithelium. "From this lymph passes by an unknown route, but ultimately reaches submucous lymph channels in the posterior region of the nose, which join together and run as submucous lymphatics beneath the posterior pillar of the fauces to the sinus pyriformis." . . . These channels can be demonstrated in certain states of inflammation of the nasal mucous membrane, if this is painted with a colored dye which will be absorbed into the lymphatics and recognized as it passes in the submucous vessels. Beneath the epithelium of the nasal mucous membrane, a small quantity of lymphoid tissue can be recognized. This tissue increases when the nose is infected by

the type of micro-organism which causes enlargement of the lymphatic glands in any other situation of the body, but does not enlarge when the nose is infected by micro-organisms which do not cause enlargement of the lymphatic glands elsewhere." The lymphatics of the antrum pass out through the ostium and along the upper surface of the inferior concha backwards on to the lateral wall of the nasopharynx.[12]

FUNCTION OF UPPER RESPIRATORY TRACT.

Yates[13] states: "The passage of the air current through the middle meatus causes the closure of the mouth and the maintenance of nasal breathing. If, owing to the deformity of the inspiratory airway, the inspiratory current passes mainly to the expiratory portion of the nose, this reflex does not take place, and, however wide the nasal airway, the patient tends to be a mouth-breather. This reflex is investigated easily by causing the patients to inhale through the nose a white powder, such as bismuth oxycarbonate, which will map out the inspiratory airway."

"Cilia: These form the motor mechanism of the nose and sinuses, and by their action mucus is conveyed towards the opening of the esophagus. The cilia form the only means of removing the secretions from the nasal air sinuses or from the middle ear. In the middle meatus the area of cilia, compared with the area of the secreting surface of the mucous glands, is roughly as 1:10. In the nasal sinuses and middle ear the area of cilia, compared with that of the secreting surface of the mucous glands, is enormously increased, this proportion being something like 50 times greater than that within the middle meatus. Of even higher efficiency is the ciliary mechanism of the bronchi and trachea."[14] This greater proportion of secreting to ciliary surface may explain the greater tendency to polyp formation. "Slight stimulation of the nasal mucous membrane causes an increase of secretion and of ciliary action."[15] Scheff[16] (1894-1895) showed by experiments on the cadaver that the air stream in respiration passes especially through the middle nasal channels. Perwitzschky,[17] in 1927, found that inspired room air at 25° C. and 35 per cent relative humidity attains a temperature of 32° C. and 79 per cent humidity in the nasopharynx during nasal respiration. Artificial dilatation of the nasal cavity by cocaine-adrenalin or contraction by swelling of the mucous

membrane after the administration of potassium iodide showed no definite differences in results from those from normal patients, although in ozena, heating of inspired air is somewhat diminished (changed anatomic structures and passages).

The Cruickshanks[18] state that "the first barrier to the entrance of organisms during inspiration is the filter of moist vibrissae (absent in childhood) lining the entrance to the anterior nares. Dust particles, bacteria and other particulate matter are caught and deposited in the vestibule, and only forcible inspiration in an atmosphere heavily charged with bacteria results in the passage of appreciable numbers of organisms into the posterior part of the nasal fossae. The projecting turbinates and the general anatomic structure of the nose ensure a rapid trapping on the moist nasal mucosa of any bacteria which may pass the first line of defenses." In 400 tests on 42 normal persons,[19] the average number of viable bacteria thus introduced was estimated at 20,000,000. From 90 to 95 per cent of the latter were rendered nonviable within five to 10 minutes. Swabs taken from the posterior pharyngeal wall showed that the bacteria are not removed to an appreciable extent by being passed into the throat from the nose. The rapid reduction of 90 per cent of the inoculated dose within five minutes did not usually hold for the hot-room environment (95° F., 90 per cent humidity). In 1896, Thomson and Hewlett[20] "found that it (ciliary passage) traveled at the rate of 25 mm. (1 inch) per minute."

In order to investigate the route by which material is removed from the various sinuses and other parts of the nasal cavities, Yates,[21] using lamp black as an indicator, observed that by the action of the ciliated epithelium the mucus was rolled over and over, thus enveloping all foreign particles. Using 0.4 per cent indigo carmine in normal saline solution at body temperature, he found that the colored fluid appeared to follow a definite path, as follows: It passed along the upper part of the middle meatus, then outwards and downwards, eventually following a definite tract in front of the Eustachian tube. From this situation it passed downwards between the attachment of the soft palate and the posterior wall of the pharynx, and, continuing, passed as a definite stream which lay just behind the posterior pillar

of the fauces. But from this part of the stream, some of the fluid passed in all cases anterior to the posterior pillar of the fauces and found its way into the recess between the tonsil and posterior pillar. At the level of the dorsum of the tongue, the main stream divided into a larger and smaller portion. The smaller portion was conveyed across the dorsum of the tongue in the position of the lingual tonsil, and thence, in some cases, it passed into the glottoepiglottidean pouch, and in other cases was swallowed directly. The larger portion continued outwards along the posterior margin of the pillar of the fauces, and thus reached the sinus pyriformis and continued down this to the posterior aspect of the arytenoids, finding its way into the interarytenoid space.

Yates[22] found that: "When placed in the posterior ethmoidal sinus, the fluid passed into the superior meatus, and escaped from this into the nasopharynx, and was conveyed outwards a short distance until it occupied a position at the upper part of the nasopharynx, immediately in front of the uppermost part of the Eustachian tube. Here it divided into two streams—a larger, which passed downwards and joined the position of the stream from the antrum, and a smaller, which passed over the Eustachian tube—and then coursed downwards through the fossa of Rosenmüller, and then curving forwards, joined the antral stream at the level of the lower border of the soft palate. If the fluid was injected into the sphenoidal sinus, it passed from the opening of the sinus in a downward direction until it reached the highest point of the posterior choanae. Here it turned backwards, and, spreading out like a fan upon the vault of the nasopharynx, was conveyed in a series of streams, the outer of which made its way into the lower part of the fossa of Rosenmüller. The inner streams passed in an outward direction until they joined together in the position of the antral stream, about one-half inch below the lower margin of the soft palate, and the further course of the fluid was as that described for the antrum. Inflammation materially slows up this process as the result of ciliary paresis or paralysis and permits bacterial proliferation with absorption of toxins."

PATHOLOGICAL FUNCTION AND STRUCTURE.

Consideration of one of the most frequent evidences of the common cold (that is, the secretion of mucus) which

manifests itself to the patient, either in the form of an anteriorly expelled discharge or as a postnasal drip, results in the obvious conclusion that this mucus secretion is the result of irritation to the mucous membrane. The most common type of irritation to the nasal mucous membrane is summarized in the word "contact." The contact, which ordinarily is not found when the nasal structures are normal, may be between the middle turbinate and the septum, the lower turbinate and the septum, the lower and middle turbinates, the turbinates or the septum and outer wall of the nose. In any case, even though it may cause sufficient irritation to produce a secretion of mucus apparent to the patient or the doctor, or both, such contact is not necessarily constant. It varies with the thermostatic variations of the nasal structures as the result of climatic or atmospheric conditions, such as chilling, overheating, excessive dryness or excessive humidity. If the combination of factors is sufficiently favorable for bacterial invasion colds result, with the train of subsequent symptoms usually manifested.

Continuous and contiguous extensions of the infectious factor from turbinate into sinus openings is the most common cause of sinus infection. Interference with sinus drainage may lead to chronic sinusitis. Improved sinus drainage by turbinate, septal shrinkage and removal of contact usually permits, in a large percentage of cases, the return of infected sinus tissue to a normal condition. If polypoid formation has occurred these tissues need treatment in addition to turbinate, septal shrinkage. Only infrequently are antral or sinus operative procedures necessary for clearing up such conditions.

Briefly, sinus infection is usually the result of turbinate, septal infection, and sinus therapeusis is dependent upon turbinate, septal therapeusis. Constant nasal or postnasal secretion, while affected by climate, need not be of wide prevalence, for a great many such cases can be largely remedied by proper turbinate, septal attention; *i.e.,* breaking the contact (through proper shrinkage) between turbinate and septum or outer wall, or turbinate and turbinate, or septum and outer wall. During the past eight years, and especially in the last three years, a sufficient number of cases have been observed for the eventual effects of the correction of these pathologico-

anatomical structures, by newer methods of high frequency turbinate, septal and lymphoid tissue treatment, to justify the conclusion that the incidence of colds in these cases has been reduced by 60 to 80 per cent. As the result of such treatment a larger air passage is obtained into areas that have not received such aeration for some time previously and there is a greater tendency to colds in the first year after treatment. During cold weather, therefore, protection by local spraying or the instillation of grease or oil should be instituted. At no time during the first year should swimming be permitted, so that chilling of the sensitive nasal mucous membrane may be avoided. These precautions, observed until the tissues have adjusted themselves to their proper function, are as a rule sufficient to eliminate this greater susceptibility to colds during the first year.

In general, the proper care of turbinates and septum by means of high frequency treatments, especially in the case of the middle turbinates, has been found necessary to eliminate a great deal of adenoidal and postnasal infection, even in cases where adenoids had been removed previously more than once by curettage. In our experience, such treatment has also served to reduce middle ear infections to a minimum. The cessation of middle ear infection after proper high frequency treatments of the nasal and throat tissues is often very striking. Inflamed retropharyngeal structures, both lateral and posterior, have been improved to a marked degree, especially as regards acute exacerbations in these areas.

My own observations indicate that, when already chronically infected from any previous cause, the lymphoid masses and lymphoid-bearing tissues in this area are very quickly flared up during the course of a cold.

The posterolateral pharyngeal columns often have sufficient extensive infection of the lymphoid, as well as the muscular structures, to demand direct therapeutic measures in addition to "shutting off the spigot from above" (the irritated nasal structures). Such attention is more easily and much more effectively given by high frequency treatment than by surgical procedures. Whether this reduction in susceptibility to colds by these means is a lessening of a negative immunological factor or the building up of an actual immu-

nity on the positive side cannot be stated at present. Attempts at elimination of the bacterial or virus factors and possible immunization by vaccine or antibodies should not be neglected, as this may build up actual positive immunity in the patient after the above treatments are carried out.

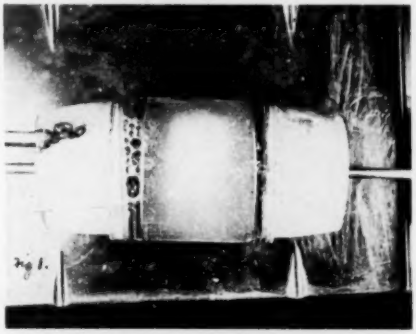
FUNDAMENTAL ACTION OF HIGH FREQUENCY ALTERNATING CURRENT IN COLLOIDS, ITS ACTION ON HUMAN TISSUES, AND ITS EFFECTS ON THE REPEATED COMMON COLD.

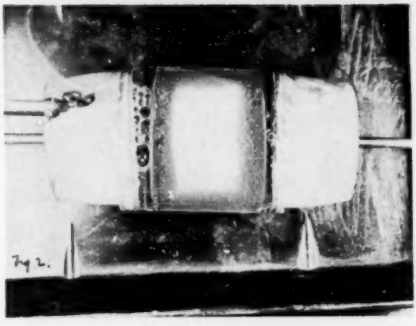
From the foregoing consideration and conclusion, one may readily see why the ordinary removal of tonsils and adenoids does not have a materially beneficial effect on the incidence of colds. Observation over a period of years has convinced us that attempts to remove lymphatic structures, such as tonsils and adenoids, by the usual instrumental means fails to completely eradicate these structures in at least three out of four cases, and, if reinfection occurs, destruction of the remaining tissues by other measures is indicated. Heat is the most formidable and effective means available for such destruction, and the selective action of high frequency current in various types of tissue has been found from practical experience to be the most effective form of heat for this purpose.

High frequency alternating current of various types can be employed in the treatment of the majority of the upper respiratory tract conditions discussed.

Experiments on the fundamental action of high frequency current have revealed differences from what might be expected on a theoretical basis.[23] As a matter of fact, no absolute proof of this fundamental action is provided us. As the result of many trials, it has been found that egg albumen is best suited for the demonstration of the action of this current. When two copper disc electrodes of equal size are employed with high frequency current in a pyrex cylinder, illustrated as in Fig. 1, coagulation first occurs in a small area midway between the electrodes. The coagulation subsequently extends into space (see Figs. 2, 3 and 4) long before the electrodes themselves become covered with coagulum. Since the center of this coagulated area is much hotter than

any other portion of the medium, steam bubbles are generated, as shown in Fig. 5, while the portion next to the electrodes is fairly solid, of the consistency of a poached egg. The central area of coagulation thus bulges during the passage of the current, until finally the electrodes themselves are also coated with solid egg white.



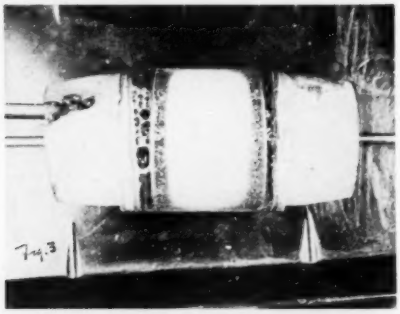


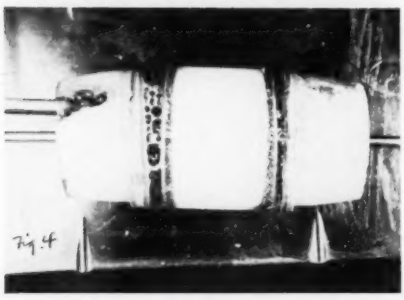
Section through this cylinder after the current has acted reveals the preceding stages of its passage. This occurs with the machine whose peak of frequency is between 800,000 and 1,250,000 oscillations, with the average voltage and milliamperage ordinarily found in the diathermy machine. In this experiment, using a standard five-foot lead for short-circuiting the indifferent tap and the medium voltage tap to test the current before and after the experiments:

On No. 4 primary control, voltage used was 7.68, milliamperage 3,000, watt 17.4.*

On No. 5 primary control, voltage used was 10.4, milliamperage 3,200, watt 20.8.

When a higher frequency is employed, such as is found in the machines employing coagulation and cutting current



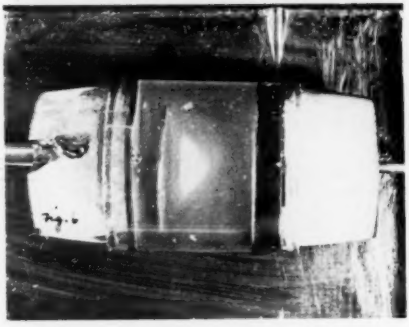


with more rapid action between the electrodes, the gradual coagulation with steaming center is replaced by a rapid solid coagulum with only a few steam bubbles centrally located. Increasing the frequency further exaggerates this same rapidly solidifying activity. Despite repeated attempts, we have been unable to obtain machines having a frequency of 200,000, 400,000 and 600,000, etc., for determination of the best fre-

*Furnished by the laboratory of the H. G. Fischer & Company on Model G-2 Diathermy, which was identical with machine used by myself.

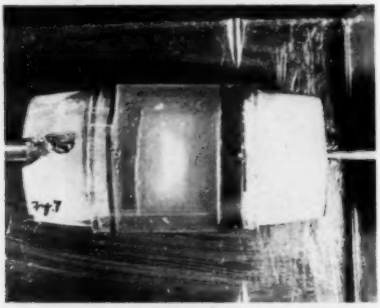
quency for this type of therapeutic work, with corresponding voltage and amperage.

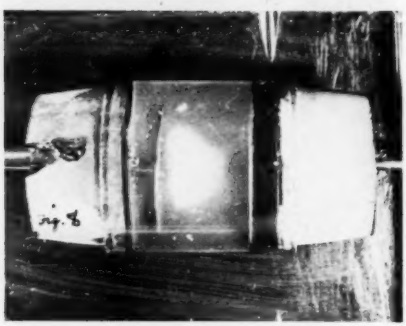
Varying the size of the electrodes shifts the center of greatest heating towards the smaller electrode (see Figs. 6, 7, 8, 9, 10, 11 and 12). This can be demonstrated diagrammatically by drawing lines from the upper edge of the smaller electrode to the lower edge of the larger, and from the upper edge of the larger to the lower edge of the smaller, the focusing effect of the heat occurring where the apices of



the two cones of current meet. In other words, the greatest point of heat under the smaller electrode begins at this focus below the surface, coagulation spreading from this area towards the larger or relatively indifferent electrode. If time is allowed, the coagulum spreads up to the actual smaller electrode and out to the larger. Decreasing the size of the electrode still further tends to focus the heat still closer to the small electrode, and when one electrode is needlepoint in size the coagulation begins immediately about it. With the proper sized electrodes, other things being equal as to

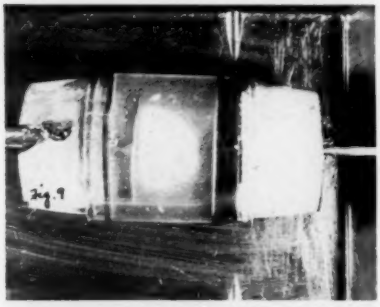
frequency, voltage and amperage, the time factor is an important one. Heat can be greatest at the focusing point and the current can be stopped so that superficial destruction is not obtained to any noticeable degree, except for slight edge effects on tissues. Or the current can be applied for a longer period of time and from the focal heat point, depending on the size of the electrode, it can be allowed to continue up to

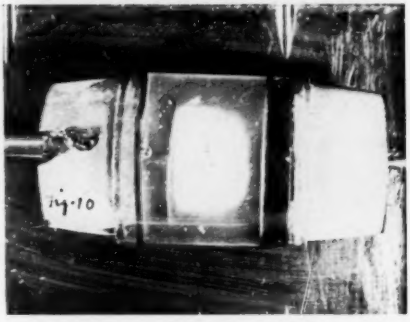




the "active" electrode or outwards to some extent towards the indifferent electrode so that an entire block of coagulated or destroyed tissue can be obtained. As the result of experiments on living and dead tissue, the 1.5 to 2 mm. ball electrodes have thus far been found most practical for giving most satisfactory effects at the anterior end of the middle turbinate, producing the proper degree of heating for shrinkage of these structures. It is applied for only one, two or

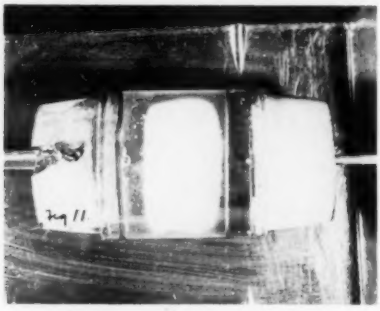
three seconds with 2,900 to 3,400 m.a., using medium voltage tap No. 4, watt 20.8. With this same voltage and milliamperage, employing a 3 x 9 mm. electrode, one obtains in three to five seconds the proper degree of cooking in the lower turbinates for the beneficial shrinkage of these structures (two to three seconds with the 3 x 6 mm., one to two seconds with the $2\frac{1}{2}$ x $2\frac{1}{2}$ mm.), which is the most widely

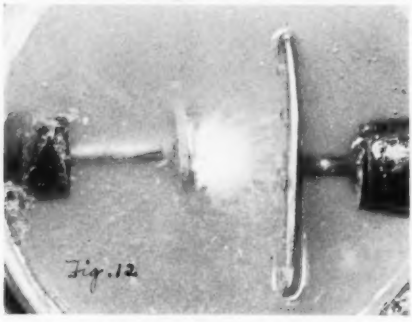




used electrode. It has been found much more effective than the various techniques which make use of standard-sized electrodes of from two to three times the surface areas mentioned over a longer period of time. The proper amount of voltage and amperage necessary to produce the same desired result for such large areas, within the tolerance period of a human being, would demand greater capacity than most of the diathermy machines are capable of registering at the present time.

The author realizes that when tests were formerly made of the effects of various frequencies, they were employed either for actual destructive purposes (true coagulation) or for medical diathermy, and, for the frequencies tested, were just as effective in one division as another. This does not hold true for the purposes discussed here. In the upper respiratory tract, between 2,900 and 3,400 m.a. are employed

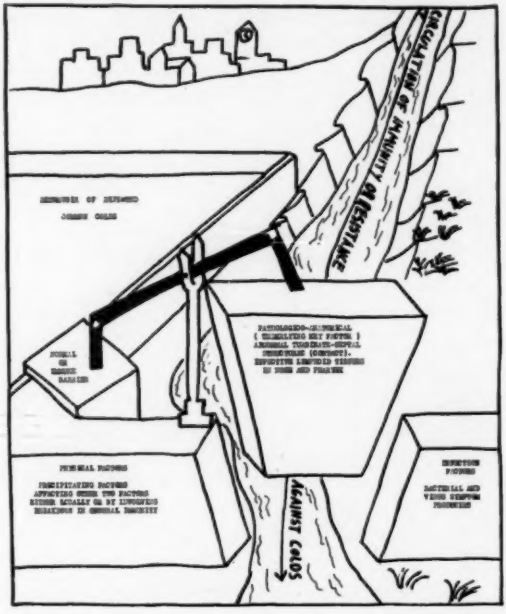




with the medium voltage tap and rheostat at No. 5, employing the ball or point electrode for destruction of tonsillar remnants and lymphatic tissues, such as retropharyngeal lymphatic follicles; or for the lymphoid tissues in the posterolateral columns, without destruction of the muscle of the pharynx.

Experiments on rabbit turbinates revealed that within the limits of the machines employed, with the sized electrodes

employed ($1\frac{1}{2}$ to $2\frac{1}{2}$ mm. ball up to 3 x 9 mm. flat electrodes) and the amount of current and time as indicated, there was either no evident destruction of tissues in them, or, when destruction of ciliary epithelium (which is comparatively resistant) occurred, it was not very noticeable. Ciliary epithelium itself is fairly rapidly regenerated, even in human beings. Mollison[24] states that: "Ciliated epithelium regenerates well in any part of the body, and the antrum is no



exception." Regeneration has been noted in man nine weeks after operation, when much of the lining had apparently been removed. One does not obtain with these methods the dry, scabby condition which usually follows instrumental attempts at correction in the upper respiratory tract. Use of the $2\frac{1}{2}$ x $2\frac{1}{2}$ mm. ball or flat electrode for the septum, middle turbinates or lower turbinates, and the $2\frac{1}{2}$ x $2\frac{1}{2}$ up to the 3 x 9 electrode on lower turbinates results in partial cooking to the proper degree, with slight coagulation, due

to the edge effects of the current in these areas. Edge coagulation is not sufficient to produce the true destructive effects that frequently follow instrumental interference.

SUMMARY AND CONCLUSIONS.

The arch of etiological factors in the common cold is composed of: *1.* The physical or the apparently precipitating factor locally upon the respiratory tract, possibly affecting the local or the general immunity. *2.* The infection factor, which is the symptom producer and bears a relationship to the individual local and general resistance or immunological condition. *3.* The keystone, the pathologicoanatomical factor of the upper respiratory tract (anatomical irritative and infective tissue) which appears to be the underlying local cause for the infection factor.

Freedom from the common cold may be represented as the circulatory stream of immunity in which the dam is the normal, immune or impermeable barrier of the reservoir of infection, while the recurrent common cold is represented by a permeable or diffusive dam of the reservoir of infection, the keystone of which is the pathologicoanatomical factor in the upper respiratory tract. This pathologicoanatomical factor is either suspended or effectively dropped into place by the lever of the normal or immune barrier attached to the fulcrum of the physical factors, as indicated at the beginning of this article. Relatively permanent freedom from or susceptibility to repeated common colds depends upon whether permanent and complete destruction of this keystone is accomplished or not (normal or immune barrier=relative freedom from colds, pathologicoanatomical factors=relative susceptibility to colds).

Elimination of the pathologicoanatomical factor in the upper respiratory tract apparently results in the effective permanent elimination of the keystone of the arch of etiological factors of common colds and provides an apparently definite means of decreasing the susceptibility to colds or increasing resistance to them, or both. The elimination of this etiological factor necessitates the usual surgical procedures only in a few cases, chiefly in young children and in a very small percentage of adults. It is best accomplished

by means of high frequency alternating current, properly applied, as previously discussed and described.

I wish to acknowledge my grateful indebtedness to Dr. Harry Homer and Messrs. Frank Martin and Wilbur Rittershofer for their valuable assistance in connection with the electrical and photographic work herein described.

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HERPES ZOSTER OTICUS.

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It is intended in this paper to present a somewhat complete picture of the interesting and rather uncommon clinical entity designated by the neurologist, J. Ramsay Hunt,[17,18,19,20,21] as herpes zoster oticus, and sometimes referred to as Hunt's syndrome.

Herpes zoster has long been known as an independent disease. It was the zona of the Greeks, and the cingula of the Romans. It has been known as shingles by English-speaking lay people for generations. Brown[2] says the first good description of herpes zoster appeared in medical literature in 1835. In 1863, von Bärensprung first showed that the lesion lies in the sensory ganglia on the posterior spinal roots. In the broadest sense, zona may be due to an acute or chronic meningitis, tabes, Pott's disease, carcinoma of the vertebrae, acute infectious diseases, intoxications, or lesions involving the posterior root ganglia, and in these cases constitutes a symptomatic zona. However, the term herpes zoster usually implies a specific infectious disease involving the ganglia of the posterior roots. Brown says that the term herpes zoster oticus was first used by Koerner, in 1904, and Gradenigo, in 1907, to refer to zoster of the geniculate ganglion. J. Ramsay Hunt collected a large number of cases involving the ganglia of the head and established zoster of the geniculate ganglion as a clinical entity.

Hunt stated that the diagnosis of herpes zoster of the auricle was made twice among 47,000 cases seen at the Manhattan Eye and Ear Hospital over a period of 10 years. The diagnosis was made once in 15,000 cases seen at the Brooklyn Eye and Ear Hospital over a period of five years. At the New York Eye and Ear Infirmary, 10,000 patients were seen yearly and the diagnosis was made six times. In a 10-year period 65,000 patients were seen at the Massachusetts Eye and Ear Infirmary and herpes zoster oticus was diagnosed 33 times. Hill[15] reported that during the last

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10 years the diagnosis was not made at the Manhattan Eye, Ear and Throat Hospital, while six cases were seen during that interval at both the New York and Manhattan Eye and Ear Infirmaries. Hunt is the only one who has collected a large series of cases, and the average case report in the literature covers only two or three cases.

It has been well established by Levaditi,[26] Olitsky,[30] Teague and Goodpasture,[36] and Lipschütz[27] that herpes zoster is caused by a filtrable virus. Goodpasture and Lipschütz produced a herpes zoster in animals by inoculation experiments. They believe that the viruses of zoster and herpes simplex differ only in virulence. Olitsky believes that the diseases are caused by separate viruses.

Fenton[10] says herpes material inoculated into the meninges of experimental animals causes encephalitic changes, but no herpetic manifestations. Olitsky has repeatedly demonstrated that the neurotropic action of encephalitis virus can be changed into a dermatropic action by subdermal inoculation in animals. Stomatitis virus can be made neurotropic and it will pass readily from the nasal mucosa of mice to the meninges and brain.

Stewart[35] believes herpes and herpes zoster are separate clinical entities, differing in the localization of the virus, their manifestations, and in results in experimental inoculation of the vesicle contents. Zoster affects the spinal type of ganglion and primary sensory neurone, and is neurotropic. Herpes is dermatropic, for it affects primarily the skin, with only incidental sensory nerve involvement. Inoculation of the contents of a herpes vesicle into the rabbit cornea gives a keratitis and no immunity. Levaditi obtained no keratitis following similar inoculation from zoster vesicles. The viruses of zoster and herpes simplex probably live commonly in the nasopharynx. The zoster virus is thought to penetrate the capillary endothelium and thus enter the blood stream from the nasopharynx and causes a transient septicemia. The virus settles in the nerve axis and spreads by the perineural lymphatics toward the center, and to a less degree toward the periphery. The virus may also spread centrally from a lesion at the peripheral nerve-ending, as does tetanus. Lipschütz believes that there is an intranuclear

proliferation of zoster virus in the spinal type ganglia and has observed characteristic intranuclear inclusions in these cases. It is known that the viruses of anterior poliomyelitis, zoster, epidemic encephalitis and simple zoster are related, but the exact relationships have not become known.

In 1904, Harvey Cushing[4] published his very carefully done and still generally accepted work on the areas of anesthesia resulting from section of the proximal roots of the Gasserian ganglion and the upper cervical roots. He showed that the line of anesthesia in the mouth following operation for tic douloureux corresponds exactly with the midline of the lip, frenum, tip and dorsum of the tongue as far back as a point slightly anterior to the foramen cecum; thence it passes outward along the circumvallate papillae to the lateral root of the tongue, where, in the majority of cases, it passes along the palatoglossus fold to the upper portion of the anterior pillar, which it follows to the uvula. The posterior pillar, tonsil and lower portion of the anterior pillar develop posterior to the mandibular arch and are therefore supplied by the glossopharyngeal nerve. The line curves around the soft palate near its junction with the uvula, gains the roof of the velum, across which it passes to the lateral pharyngeal wall. It passes upwards, dividing the pharyngeal ostium of the Eustachian tube, thence to the midline of the vault of the pharynx; from here it pursues again a median path along the intranasal septum, keeping in the midline of the soft palate and uvula, both dorsal and ventral surfaces, hard palate, gums and upper lip to the mucocutaneous junction. The area of cutaneous anesthesia produced by section of the Vth cranial nerve roughly covers the lateral surface of the face from the midline to the anterior wall of the external auditory canal and anterior half of the tympanic membrane, and from the forehead to the ramus of the mandible. He pointed out that the concha and posterior wall of the external auditory canal lie between the cervical field posterior and the trigeminal field anterior, and went on to say that it was generally believed by anatomists to be supplied by the aberrant cutaneous branch from the vagus, the R. auricularis nervi vagi of Arnold. He also expressed the belief that there was clinical evidence to show that both common and special sense fibres pass from the tongue through the chorda tympani and N. intermedius.

It was J. Ramsay Hunt who did most to establish the peripheral distribution of the sensory root of the VIIth cranial nerve. In the light of embryological studies the VIIth, IXth and Xth nerves are to be regarded as branchial nerves, which in the lower vertebrates stand in immediate relation to their respective visceral clefts and arches. The VIIth is the nerve of the first visceral cleft and in lower forms the sensory portion is much larger and in addition to a cutaneous distribution is probably distributed also to the lateral wall of the pharynx. From the first cleft and its adjacent arches are developed the structures of the external ear, the tympanic membrane closing and separating it from the first visceral pouch. The anterior or mandibular arch lies within the area of the trigeminus, and gives rise to the tragus and the crura of the antihelix of the adult ear. The posterior, or hyoid arch, is innervated by the facial system. From this arch are developed the auricular tubercles of the antitragus, antihelix and the lobule. As the concha auris and the external auditory canal are similarly related to the first visceral cleft, they, too, may be regarded as falling in part within the area of the innervation by the VIIth nerve. The auricular branch of the vagus is considered by some embryologists to be a remnant of the so-called lateral line, and has been traced as far forward as the first visceral cleft, where, as dissection studies show, it is partly distributed. Embryological studies show that the tragus and crura of the antihelix are situated within the area of the trigeminus; the antitragus, lobule, antihelix, concha and part of the auditory canal lie within the area of the facial; and the auricular branch of the vagus may be traced into the first visceral cleft.

From the standpoint of gross anatomy we know the auditory nerve passes with the facial nerve and intermedius nerve into the internal acoustic meatus and divides into the cochlear and vestibular nerves. Along the vestibular nerve are distributed the ganglion cells of Scarpa. The cochlear nerve is distributed to the modiolus and bears the spiral ganglia. The intermedius nerve lies close to the facial, and forms the geniculate ganglion at the genu of the facial nerve just as it leaves the Fallopian canal to enter the tympanic cavity. The geniculate ganglion has connections with the sphenopalatine ganglion through the great superficial petrosal

nerve, and with the otic ganglion through the small superficial petrosal nerve. The fibres of the intermedius nerve are distributed in part to the chorda tympani. In cases of facial palsy, the general sensation of the anterior two-thirds of the tongue may be decreased. Hence the nerve probably carries some general sensation. The lingual branch of the facial nerve innervates the stylohyoid and palatoglossus muscles at the base of the tongue and sends fibres to the anterior faucial pillars. The geniculate ganglion may then have a vestigial intraoral sensory representation in addition to its taste function.

Hunt summarizes the nerve supply of the auricle as follows: the Gasserian ganglion is represented by auricular filaments, which arise from the auriculotemporal branch of the third division; the geniculate by auricular fibres, which emerge with the facial at the stylomastoid foramen, the exact course and distribution of which is not certain; the petrous ganglion of the glossopharyngeal nerve and the jugular ganglion of the vagus by their respective auricular branches; and the second and third cervical ganglia by the auricular branches of the cervical plexus. Clinical and dissection studies show that the Vth nerve is concerned in the innervation of the anterior portion of the tympanic membrane, external auditory canal and meatus; and the vagus in the innervation of the posterior portion of the tympanic membrane and auditory canal, together with a strip on the posteromesial surface of the auricle. The cervical nerves supply the lateral surface of the neck, the mesial surface of the auricle, and are represented on the lateral surface of the auricle over the helix, the fossa of the helix and lobule. These distributions leave the concha, tip of the antitragus, a portion of the antihelix and fossa of the antihelix still unaccounted for, and the innervation of this area is believed to be related in part to the geniculate ganglion of the VIIth cranial nerve.

The glossopharyngeal nerve is distributed to the mucous membrane covering the posterior margin of the soft palate, the anterior and posterior pillars of the pharynx, the tonsil, the posterolateral surface of the base of the tongue, and to the pharyngeal sympathetic plexus. The sensory filaments from the vagus nerve pass to the mucous membrane covering the epiglottis, arytenoid eminences, arytenoepiglottic

folds, a wedge-shaped area at the base of the tongue, situated immediately in front of the epiglottis, and sends branches to the pharyngeal plexus.

An understanding of the histological similarity between the spinal root, geniculate, spiral and cochlear ganglia and those of the glossopharyngeal and vagus nerves is essential to a conception of the zoster syndrome as described by Hunt. All these ganglia take their origin from the neural crest. In lower forms the cells of these ganglia are all bipolar, and in the course of phylogeny become unipolar except in the ganglia of the cochlear and vestibular branches of the VIIIth nerve, where the cells retain the bipolar characteristic even in man. Hunt conceives of these ganglia of the spinal nerves, the Vth, VIIth, IXth and Xth cranial nerves as forming a continuous chain with similar architecture. It would be but logical to expect that they would show the same response to specific inflammation.

The skin lesion of zoster, the vesicle, resembles that of smallpox, but contains more exudate. It is due to intercellular edema, with local necrosis, and the appearance of Unna's "balloon cells," large, swollen and multinuclear. Late skin changes include a thickened stratum corneum and proliferation of pigment cells, with depressed fibrotic zones when secondary infection has occurred.

Dabney[5] writes that injection, hemorrhage, infiltration and degenerative changes in the posterior root ganglia in zoster were reported by Charcot, Cotard, Wyss, Sattler and Dubler before the classical work of Head and Campbell, in 1900.

The following description of the histopathology is taken from Head and Campbell.[16] If the patient dies with the skin manifestations still present, there will be clumps of round-cells spread through the interstitial tissue of the ganglion involved. These foci are commonly located about extravasated blood. In some cases the ganglionic structure will be more or less destroyed. In the center of the hemorrhagic areas the ganglion cells are entirely destroyed eventually. Over that portion of the ganglion which is inflamed the sheath generally shows similar changes. The greater the severity of the eruption, the more profound the scarring that is left behind. When the inflammation has spent itself, absorption

sets in. The effused blood, if not entirely absorbed, remains as a brownish granular deposit in the center of the scar tissue. The final result is usually a scar occupying one-sixth to one-half of the ganglion. Within this area the ganglion cells and nerve fibres are destroyed, and no structure remains to show where they once existed. In most cases, some functioning ganglion cells and nerve fibres remain. Over the sclerosed patch in the ganglion the sheath becomes converted into an almost homogeneous mass of dense fibrous tissue.

The changes in the posterior nerve root correspond to the lesions in the ganglion. There is an acute degeneration, followed by more or less secondary sclerosis, according to the severity of the acute degeneration. The anterior root is always normal. An acute degeneration of nerve fibres leading from the area of skin involvement to the involved ganglia usually appears about the tenth day of the disease, and resolves or goes on to scar tissue formation, depending upon the severity of involvement of the posterior root ganglion.

Acute degeneration takes place in the fibres from the involved ganglia which ascend and descend in the spinal cord. This acute degeneration begins in nine or 10 days after the onset of the ganglionitis and the process results in complete absorption without scar formation.

Head and Campbell reviewed cases which suggest that zoster in all respects similar to that arising spontaneously may be produced by implication of a posterior root ganglion in inflammatory processes secondary to malignancy, tuberculosis or injury to the spine.

Herpes zoster oticus is a self-limited disease, begins with a vague prodromal period, passes through a more or less severe course covering a week or two, and gradually leaves, usually without permanent sequelae. The prodromal stage usually lasts from two to five days and manifests itself by malaise, mild gastrointestinal upset, occasionally fever, and localized hyperesthesia which develops into a pain. Pain is the most constant and distressing symptom. It is deep-seated in the ear, and may be radiating, pulsating, lancinating, boring or prostrating. Within a day or two after the onset of the severe pain the external auditory canal and auricle become indurated and edematous, and characteristic herpetic

vesicles develop in the concha, over the antitragus, posterior canal wall and, occasionally, the tympanic membrane itself. With this involvement of the external auditory canal, some degree of deafness, conductive in type, develops. The severe pain continues unabated. The vesicular eruption may remain for weeks, but usually clears in 10 to 14 days, leaving tiny pigmented scars. The herpetic pain then gradually subsides, but bothersome hyperesthesia may persist for six months or more after the disappearance of the eruption. A complete, peripheral facial palsy usually develops a few days after the appearance of the eruption; but it is not invariably present, and is not needed to make the diagnosis. Such is the clinical picture and course in a mild zoster involvement limited to the geniculate ganglion. In a severe involvement, facial paralysis is probably always seen. In the more severe degree of involvement, a perceptive type of deafness develops due to infiltration of the spiral ganglia. When the vestibular ganglia are affected, the symptomatology may vary from mild vertigo to severe manifestations of Méniére's symptom complex.

The symptoms and findings in zoster involvement of the ganglia of the glossopharyngeal and vagus nerves can be predicted from the anatomical study presented. Here there may be a herpetic eruption in the concha and external auditory canal, but the eruption is always present on the posterior and lateral wall of the pharynx, over the aryepiglottic fold and epiglottis. Dan McKenzie[28] reported an interesting case of multiple ganglion involvement. Here herpetic lesions developed in the fossa of the helix, on the scalp above and posterior to the auricle, right malar eminence and upper lip near the right angle of the mouth, the right edge of the tongue, right anterior pillar, tonsil, posterior pillar, right lateral retrotonsillar pharyngeal wall, right half of the posterior pharyngeal wall, right edge of the epiglottis, upper aspect of the right aryepiglottic fold and right arytenoid. The nerve involvement included the right facial, weakness of the right sternomastoid, the upper fibres of the right trapezius muscles and the right recurrent laryngeal nerve. The ganglia involved probably included those of the VIIth, IXth, Xth, XIth and Vth. McKenzie believes that the recurrent laryngeal nerve paralysis is often transitory and prob-

ably missed. Involvement of the peripheral vagus nuclei may also produce varying degrees of nausea and vomiting.

Ramsay Hunt's classification of cephalic herpes zoster depends largely upon the degree of severity and multiplicity of the ganglionic involvement. He gives as divisions: *1.* Herpes oticus; *2.* herpes oticus with facial paralysis; *3.* herpes oticus with facial paralysis and hypoacusis; and *4.* herpes oticus with facial paralysis and Ménière's symptom complex. As subgroups he adds: *1.* Herpes facialis with facial paralysis and auditory symptoms; *2.* herpes occipitocollaris with facial palsy and acoustic symptoms; and *3.* herpes of the cephalic extremity with auditory complications.

A workable concept of the symptomatology of herpes zoster oticus can be based on an understanding of the neural anatomy and pathology of the condition. The condition can vary from herpes oticus without facial paralysis to a severe involvement of many cranial nerve ganglia with parallel increase in symptoms and local manifestations. Because of the varying extent of ganglionic involvement which may present itself, a multidimensional picture of the condition is essential.

If herpes zoster is kept in mind, the diagnosis should always be readily made by the otologist and its differentiation from simulating conditions should not be difficult. A complement fixation test utilizing serum from a person who has recovered from herpes zoster has been suggested, but not widely used. The diffuse and painful swelling over the mastoid, concha, posterior wall of the external auditory canal, prior to the appearance of herpetic vesicles, or after such vesicles have dried up or coalesced into open ulcers, must be distinguished carefully from other causes of similarly painful edema. Subperiosteal edema from mastoid disease has deep tenderness on pressure and a fairly characteristic Roentgenogram. Edema from furunculosis has painful swelling and pain on traction on the pinna. Eczema and pyoderma lack the severe neuralgic pain of zona. Erysipelas has a higher temperature and a raised, brawny border. Fungus infections produce no vesicles and do not have the neuralgic pain. Facial paralysis with otalgia and a negative middle ear should always suggest zoster. After head trauma and skull fracture, hemor-

rhage about the internal auditory meatus or into the tympanum may cause palsy, and tension on the dura over the Gasserian fossa may produce severe pain. Lesions of the VIIth nerve from cerebellopontine angle tumors or cysts produce auditory, vestibular and ocular changes which are slowly progressive. Boring or band-like headache, often with transitory rise in temperature and mental irritability and confusion, may suggest intracranial hemorrhage in hypertension, thrombosis in hypotension, the prodromal stage of epidemic cerebrospinal meningitis or encephalitis, and invasion or extension by brain tumor or abscess. Severe pain in the facial sensory distribution, relayed at the geniculate ganglion from the great superficial petrosal and Vidian nerves, may be caused by posterior sinusitis and rarely by irritation or malignant lesions of the nasopharynx. Neuralgias due to dental caries, use of arsenicals in root canals, impacted third molars, undescended cuspids, cysts, osseous tumors of the jaw and like conditions are easily segregated by Roentgenologic evidence and the absence of the typical skin lesions. The differential diagnosis can usually be made if the condition is kept in mind.

The prognosis for cessation of symptoms and for life is good. The eruption usually dries up in a week or two, to leave a yellow brown crusting, which may take months to disappear. The facial paralysis, auditory and vestibular manifestations usually are gone in the course of two or three months. The residual pain and hyperesthesia may persist for six months. Hunt and Muck[29] reported cases where deafness and facial paralysis persisted. Jaehne[22] saw one case in which permanent unilateral loss of sense of taste was present, with deafness and facial paralysis. Recurrence is rarely seen, but Kaposi[25] reported a case with nine recurrences, and Politzer[31] one with three recurrences.

The management of herpes zoster oticus is symptomatic. Dry open treatment with nonirritating powders or mild, quickly drying antiseptics will obviate the development of secondary infection in the vesicles. Ample use of hypnotics and narcotics is usually necessary to control the pain. Fenton says cocainization of the sphenopalatine ganglion often reduces the pain and vertigo. Foreign protein therapy and autohemotherapy have been recommended by Rebattu[32] and

his coworkers. The serum of cured cases and diathermy may shorten the duration of the postherpetic pain. Operative measures to decompress the facial nerve should not be considered until the facial paralysis has existed unchanged for six months. Hill[15] advised trial of Roentgen ray therapy.

Herpes zoster oticus is then a self-limited clinical entity caused by a filtrable virus involving the geniculate ganglion of the facial nerve; occasionally associated with similar involvement of the ganglia of the Vth, VIIIth, IXth and Xth cranial nerves; characterized by severe otalgia, vesicular eruption in the concha, or the posterior canal wall, posterior half of the tympanic membrane, and facial paralysis; usually recovering without residual paralysis or deafness; and seldom showing recurrence.

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SENSITIVITY TO FLOWERS.*

DR. JOSEPH B. BIEDERMAN, Cincinnati.

As early as 1565, Botallus[1] reported an individual who, after smelling roses, would have a violent attack of sneezing, rhinorrhea and headache. In 1819, Bostock[2] reported the first description of hay fever. Blackley's[3] Publication, in 1873, scientifically proved the importance of pollens as a cause of hay fever.

Several general rules have been set down to distinguish those plants which are wind-pollenated from those that are insect-pollenated. The plants that are wind-pollenated give off great quantities of light pollen, which float in the air and cause trouble when they come in contact with sensitive individuals. The pollens from insect-pollenated plants, however, are too large and heavy to travel far from their original source and depend upon attachment to the bodies of insects for their transportation. In order to attract insects so that they may be fertilized, these plants are usually pretty, due to various gay colors, or have a pleasant odor. Due to the large size pollens of the insect-pollenated plants it is necessary for the pollen-sensitive individual to have very close contact with these plants; for instance, smelling them, in order to develop symptoms. In this way the heavy pollens are brought into direct contact with the sensitive individual's nose.

The object of this paper is to point out the fact that people may develop allergic symptoms without being sensitive to the plant pollen. I wish to add that this article is not meant to refute the pollen theory of the causation of allergic symptoms, which is definitely established, but to call attention to the fact that the odors emanating from the flowers may also cause symptoms. In our conviction that pollens cause allergic symptoms, we have disregarded the fact that odors may also cause symptoms in susceptible individuals. In the

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past it had been the custom of general practitioners of medicine, on purely empirical grounds, to have all flowers removed from the rooms of allergic individuals. Lately, however, due to the demonstrations that the pollens of the plants are the offending agents, the physician has permitted pretty and odoriferous plants to remain in the environment of the allergic individual, smug in the knowledge that the pollens of these plants are too heavy to float in the air and that as long as the patient did not put his nose close to the flowers he was safe.

A patient came to me after being treated by a well informed allergist, who did not get results from treatment simply because the patient was permitted to have insect-pollenated plants about the home. In this patient the asthma cleared up when the flowers were disposed of.

To determine whether or not the odor from plants, as well as pollens, can cause allergic symptoms, the following experiments were performed: Miss A. S., a school teacher, age 40 years, who has rose fever, told me that the odor of cosmos and marigolds made her have a nasal discharge of a thin watery nature and also caused her to sneeze from 10 to 20 times in succession. Her past and family histories were negative for allergic manifestations. Her physical examination revealed nothing abnormal. Intradermal skin tests with both cosmos and marigolds, in 1-100 dilution, were negative. Passive transfer tests to these pollens were also negative. Cosmos and then marigold pollens placed in the nostrils and the eyes of this patient caused no reaction. Timothy grass pollen, however, in 1-1,000 dilution, was positive on intradermal and passive transfer tests. Some marigolds were then placed in a room and the patient told to sit in the hall next to this room. A vaseline-covered slide was held adjacent to each nostril. Within a few minutes after the patient had seated herself in the hall she began to sneeze, her nose became clogged and had a thin watery discharge coming from it. The vaseline-covered slides adjacent to her nostrils were free from marigold pollen. The exact findings were again recorded after the same experiment was repeated at a subsequent date, with the exception that cosmos was substituted for the marigolds.

Mrs. F. R., housewife, age 42 years, complained of asthma and hay fever from about Aug. 15 until the first frost. She volunteered the information that an attack of asthma was sure to follow if she smelled a marigold. Her father and mother both had asthma, and she had eczema as a child. Her physical examination was entirely normal. Skin testing revealed her to be sensitive to ragweeds in 1-10,000 dilution, cocklebur and wormwood in 1-1,000 dilution, and negative among other tests to the marigold pollen in 1-100 dilution when tested intradermally. The marigold pollen when placed in her nose and eyes gave negative reactions. Passive transfer tests were negative with marigold pollen, but positive with ragweed, cocklebur and wormwood pollens. The same experiment was repeated as with the aforementioned patient. Marigolds placed in an adjacent room to this patient caused her to have asthma, whereas the vaseline-covered slides beside both nostrils were free from the marigold pollen.

Mr. L. R., age 30 years, complains of rose fever present three years. His symptoms usually begin in early May and last until the first part of July. His past history revealed the presence of hives when 8 years old, which lasted two years. His family history showed that his mother had rose fever before her death. His physical findings were normal. He told me that if he stepped into a home where there were roses, he would begin to sneeze immediately, his nose would run and stop up. The flowers would not necessarily have to be in the same room to cause him to have symptoms. Rose pollen, 1-100 dilution, was negative on intradermal and passive transfer tests, and also negative when placed in his nose and eyes. Timothy, orchard grass, June grass and red top in 1-10,000 dilutions were all positive, however, on intradermal skin testing. The same experiments were repeated as with the other two patients. He also developed sneezing and rhinorrhea when placed next to a room containing roses. Vaseline-covered slides held adjacent to his nostrils at the time of exposure contained no rose pollens.

Many more patients could be cited who were definitely sensitive to the odor of other flowers, such as lilacs, gardenias, etc.; the above patients are merely cited as typical examples.

In a future communication I shall report the results of my tests on a large number of individuals, with essential oils

used to give an odor to various commercial products, such as soaps, cosmetics and other toilet articles. At the present suffice it to say that quite a few different essential oils are also capable of causing allergic symptoms in susceptible individuals.

SUMMARY.

Cases are presented to illustrate the fact that odors of plants are capable of causing allergic manifestations, even though the pollens of the same plants were not the offending agents.

COMMENT.

This paper is written to call attention to the fact that insect-pollenated plants are potential trouble-makers for allergic individuals, even though their pollens are too heavy to be carried far or very long in the air. They may cause allergic symptoms, due to their odor. In our acceptance of the pollen theory as the cause of allergic symptoms, we must not overlook the insect-pollenating plants as potential trouble-makers as a result of other properties than pollen, namely their odor.

ADDENDUM.

Since submitting this article for publication I have had the opportunity of testing the two individuals sensitive to marigold odor with the new Crown of Gold marigold that has the odorless foliage, obtained through the courtesy of the W. Atlee Burpee Co. These plants, when placed near the individuals sensitive to the odor of marigold, caused no symptoms.

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KINETICS OF STAMMERING: TWO FACTORS THAT SEEM TO FACILITATE VOICE PRODUCTION.*

DR. CLARENCE QUINAN, Nevada City, Calif.

In a study carried out during a period of a little over three months with two young women afflicted with stammering, quantitative data in regard to the number of speech errors they made were obtained by means of a plan which enabled me in each instance to determine at once what tones gave trouble and also the basic rate of the speech disorder. This plan consisted simply in noting down each articulation fault the stammerer committed while she repeated four times in succession a list of 25 selected test words. In recording the results, each mistake made in pronounciation was counted as an error, no matter how slight or how excessive the superfluous articulation effort might be—whether it amounted merely to a slight repetition-act or to complete blocking on a word along with a struggle. The scores thus secured, respectively, as to the four word panels were set down separately. It was found[1] consistently, as to six different lists of test words, that the four consecutive panel-values obtained in any given instance pinched out rapidly in the direction from left to right—*i.e.*, from panel No. 1 to panel No. 4. This kinetic phenomenon was styled the "disappearance-trend."

Although, *a priori*, it was impossible to foresee that disappearance-trends would manifest themselves, yet one could at least be sure that all panel-values noted would be quantitative ones; and it seemed likely that such values might well be useful as a yardstick with which to compare other similar data referable to any factor capable of modifying voice production. With this idea in mind, therefore, at the very outset of the experimental work done by the panel method, three kinds of data as to speech errors were collected from each person tested. These data were obtained while she was: *1.* In a state of physical repose; *2.* scrambling about on all fours; *3.* writing a model word with both hands

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at once—each hand moving in the direction away from the midline of her body.

What evidence warranted the assumption that either one or the other of these two unusual procedures or handicaps might enable a reactor to improve her score on a given test list, as shown by yardstick values?

Geniesse,[2] who studied 24 persons afflicted with stammering, states that "a marked improvement, even to a complete cessation of stuttering, was noted when the stutterer spoke while walking on all fours."

With respect to the bimanual test, on the other hand, it was known that two-handed writing movements, if performed simultaneously, produced invariably a sort of stammering activity on the part of the muscles of the upper extremities.[3] Hence, it seemed worth while to find out in what respect, if at all, the tendency to stammer might be modified if muscle movements referable to the upper extremities as well as those referable to the midline speech mechanism were performed at once.

The data obtained seemed to show that each one of the handicaps referred to facilitates voice production; but that in this respect the quadrupedal procedure was decidedly less effective than the bimanual writing test.

It is the object of this article to describe briefly the data in regard to speech errors secured, respectively, under these two handicaps, the errors noted referable to the ordinary physical status having been reported in an earlier paper.[1]

METHOD.

Since the results obtained in tests made with two stammerers agreed perfectly, I shall describe here only the findings noted as to the reactor M. S.

An account of the technique employed in the panel method of speech testing having already been published,[1] it will suffice here to add a few details about the quadrupedal test and the bimanual graphic test, respectively.

Presumably, one can "walk on all fours," either by means of the elbows and knees or by means of the fingers and toes. However, choice fell upon the latter method simply because it seemed to be the neater one. In carrying out a speech

test in this awkward fashion the one essential point to be observed is that the reactor should try to pronounce each word only while his body is actually in motion.

In practice the bimanual graphic handicap was applied by having the person being tested pronounce each word while writing with both hands at once the single test word, "Halliard." This word was written again and again as often as necessary, each hand moving in the centrifugal direction. In this test one technical point must be stressed: all muscle movements, those of the two hands and those of articulation, must be exactly synchronized.

As a rule the tests were put through by first gathering data in regard to the speech errors made habitually—these were the data subsequently used as the yardstick values—afterwards the mistakes made, respectively, under the bimanual and the quadrupedal handicaps were recorded.

I shall describe the panel data first and add thereafter a few notes as to some observations made in collateral experiments.

TABLE I.

Lists of Words Used in the Panel Tests.

1	2	3	4	5	6
Downey	Downey	Disestablish	Whittle	Mower	Zebulon
Business	Business	Domesticity	Thunder	Hemlock	Millet
Helena	Riot	Butchery	Smear	Nuncio	Gurgle
Bobby	Mother	Motherhood	Mutter	Murmur	Mammal
Jacobs	Submit	Hemisphere	Nutrition	Native	Yataghan
Vienna	Industry	Bobby	Whistle	Typical	Vehement
Tulip	Disposition	Number	Thrift	Mottle	Ultimate
Chopper	Wilson	Wilson	Shave	Whittle	Querulous
Emma	Invested	Invested	Mugger	Notation	Ribbon
Bologna	Goggle	Mrs.	Nifty	Mosquito	Omnivorous
Rogers	Production	Protuberant	Whisper	Newtown	Nullify
Figure	Mrs.	Theater	Thrive	Mullet	Hummock
Jelly	Delivering	Gutteral	Mummy	Muddle	Minimal
Garage	Theater	Nature	Nymph	Butter	Immunity
Petunia	William	Whither	Whoop	Bitter	Lullaby
Tuttle	Motion	Motion	Thyroid	Mister	Lubbock
Junket	Ruin	Ruin	Shatter	Mortar	Maple
Whippet	Gutter	Washing	Mulatto	Moulting	Numeral
Senator	Must	Wearisome	Numeral	Nickname	Vivify
Rowett	Watching	Simian	Whither	Moslem	Mummy
Bigelow	Number	Noontide	Thrips	Mumpish	Whiffle
Salmon	Supervisor	Siphon	Shamble	Skewer	Naples
Nugget	Senior	Moonlight	Multiply	Muscovite	Muffle
Canary	Nature	Rummage	Norwich	Mulberry	Money
Billy	Site	Money	Shoot	Mimeograph	Mugger

Data in regard to the speech errors made by the stammerer M. S. as determined by the panel test method. Comparison of the habitual values with those referable respectively to a quadrupedal and bimanual handicap.

		SETS OF PANEL VALUES											
		"A" Habitual				"B" Quadrupedal				"C" Bimanual			
Date	Test List	1	2	3	4	1	2	3	4	1	2	3	4
8/5	1	12	8	7	5	6	4	2	0	10	8	7	6
8/8	2	8	8	7	0	0	0	0	0	7	4	0	0
8/12	2	0	0	0	0	0	0	0	0	0	0	0	0
8/15	3	5	3	0	0	0	0	0	0	0	0	0	0
8/25	3	5	1	0	1	0	0	0	0	0	0	0	0
8/29	3	2	1	0	0	0	0	0	0	0	0	0	0
9/5	4	6	2	2	1	8	1	0	0	1	0	0	0
9/6	4	0	0	0	0	0	0	0	0	0	0	0	0
9/19	5	4	5	2	0	5	1	0	0	3	1	0	0
9/20	5	1	3	1	0	3	4	0	0	0	0	0	0
9/21	5	3	1	1	0	2	2	1	0	2	2	0	0
9/22	5	2	1	0	0	3	2	1	0	2	0	0	0
9/23	5	2	1	0	0	2	2	1	0	2	1	0	0
9/24	5	0	0	0	0	0	0	0	0	0	0	0	0
10/3	5	0	0	0	0	0	0	0	0	0	0	0	0
11/10	6	1	1	2	0	4	4	1	1	3	2	1	1
11/11	6	4	2	4	2	3	3	1	1	4	3	2	0
11/12	6	4	3	2	1	4	2	0	0	0	0	0	0
11/13	6	5	3	1	0	3	4	1	0	0	0	0	0
11/14	6	0	0	0	0	0	0	0	0	0	0	0	0
Totals		64	43	29	10	43	29	8	2	34	21	10	7

RESULTS.

On comparing the three sets of panel-values exhibited in Table 2, it will be evident at a glance: *1.* That all three display the characteristic disappearance-trend in the direction from left to right; *2.* that the respective sets of data referable to the handicaps—both "B" and "C"—are notably smaller than the yardstick values "A"; *3.* that the smallest values were those noted under the bimanual handicap.

To be exact, the relative figures obtained as to the number of speech errors made were, in percentage terms: habitual, 7.3; quadrupedal, 4.1, and bimanual, 3.6. In other words, the bimanual handicap had the effect of cutting down by about 50 per cent the habitual rate of stammering.

Both stammerers testified voluntarily that the bimanual handicap seemed to facilitate the utterance of vocal sounds; that in this respect it appeared to be decidedly more effective than the quadrupedal handicap.

Relatively speaking, however, since undoubtedly it is much easier to execute the coarse movements made in walking on all fours than it is to perform the delicate and complicated task of writing with both hands at once, it was assumed that the graphic handicap enabled the speech mechanism to function more smoothly, for the simple reason that, in comparison with quadrupedal efforts, the performance of two-handed writing tended, in a much greater degree, to distract the will or attention factor. At any rate, the available evidence, such as it was, seemed to show that the exact synchronization of the handwriting movements, right and left, a maneuver which invariably caused a sort of stammering response or sprawl phenomenon on the part of the muscles supplied by the brachial plexus, curiously enough, diminished at the same time the number of speech errors made by the stammerer M. S.

Evidence of a similar tenor, *i.e.*, as to the presence of a disappearance-trend, was derived also from other sources.

During the period of six days, from Aug. 31 to Sept. 5, 1936, a few collateral experiments were carried out in the following manner: On the first day, M. S. having been asked to read aloud from an article in a popular magazine, and the mistakes she made in pronunciation having been carefully noted and tabulated, it was thus found, in quantitative terms, that she committed 50 errors while reading a total of 654 words. On the second day, however, her errors numbered but 27, while the scores she made on the same passage during the ensuing four days were, respectively, 12, six, three and three. At first, it was noted, her lips snapped shut on the tones "M" and "N"; but within six days her speech mechanism largely overcame this difficulty, thus displaying seemingly a remarkable quality of adaptibility.

Again, however, apart from her stammering, it was obvious that M. S. had another kinetic, habit-peculiarity. This was that from time to time she touched her lips or chin with her fingertips. To correct this habit she was asked to drop a grain of rice into her purse each time her hand strayed upward to her face. Since this irksome task was too troublesome to be persisted in, M. S. scored herself thus for a period of but five days. However, during this five-day period the respective counts made of the rice grains in her purse were 164, 104, 51, 52 and 38.

Hence, various kinds of kinetic data, some referable to the midline speech mechanism, others to movements of the upper extremities, resembled one another clearly and definitely, in that all displayed disappearance-trends of one sort or another.

COMMENT.

Did the graphic discipline imposed upon the hemispheres, left and right, until their respective velocity capabilities had been integrated to the utmost modify in any way the tendency to stammer? It would seem conservative to state, I believe, that the experimental work carried out by these two stammerers was decidedly helpful in both instances, in that it seemed to help them toward a better understanding as to the basic cause of stammering. At any rate, long after the experimental work was discontinued, there can be no doubt that, in both instances, the speech disorder was markedly improved. This was commonly noted. In the case of M. S., for example, after the lapse of seven months, with no additional training whatever, she read aloud to me a passage numbering upwards of 1,300 words without committing a single speech error; indeed, she seemed to have acquired a quality of voice control hardly to be distinguished, if at all, from the normal. Now, was this very striking improvement due to the panel tests or to the graphic training process? Or was it due to such an abstract cause as the sheer impact between two unlike personality factors? These questions cannot be answered decisively with such data as are now available.

CONCLUSIONS.

1. Experimental evidence is advanced which seems to show that the use either of a quadrupedal handicap (walking on all fours) or a bimanual handicap (writing with both hands at once) greatly facilitates voice production.

2. Quantitative data of several kinds obtained from stammerers and referable to different respective levels of the cerebrospinal axis resembled one another, in that they exhibited without exception well marked disappearance-trends.

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THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The sectional meetings of The American Laryngological, Rhinological and Otological Society, Inc., are scheduled as follows:

Eastern Section, Philadelphia, Pa., Jan. 7, 1938; Southern Section, Atlanta, Ga., Jan. 24, 1938; Middle Section, St. Louis, Mo., Jan. 26, 1938; Western Section, Santa Barbara, Cal., Jan. 30-31, 1938.

Programs of these meetings will be mailed to the Fellowship in December.

Subject to confirmation by the Council, the annual meeting will be held at the Hotel Claridge, Atlantic City, N. J., April 27, 28 and 29, 1938, immediately preceding the meetings of the Congress of American Physicians and Surgeons, and the other special societies.

A scientific program of exceptional interest is being arranged, and distinguished otolaryngologists from England and France have already signified their intention to attend.

CARCINOMATOUS METAPLASIA IN A CASE OF UNCONTROLLABLE POLYPOSIS OF THE NOSE.*†

DR. IRVING B. GOLDMAN, New York.

Most authors who have considered the association of nasal polypi and malignant tumors have expressed extreme doubt concerning the possibility of the malignant transformation of the innocent benign nasal polypi. The nasal polypi are frequently found as a complication of malignant tumors is generally accepted, nevertheless there is a universal consensus of opinion that a nasal polyp does not become transformed into a malignant tumor, but that the polyp appears in certain cases as a result of a deep-seated primary malignant growth under the mucous membrane from which it arises or to which it is attached.

The following case report covers a period of 42 years and demonstrates that nasal polypi are not always benign, but that they can undergo epithelial metaplasia and become carcinomatous.

Report of a Case: The patient, a female, age 52 years, was first seen in the Mount Sinai Hospital out-patient department in 1895. At that time she complained of nasal obstruction and excessive snoring. Her mother had looked into her nose and had noted the polypi. Examination by Dr. Meierhof confirmed this observation. The polypi were removed, but grew back rather promptly. She returned weekly for polypectomies for seven years. She also visited other institutions and her sinuses were operated upon by many competent rhinologists without success.

Thirty-two years ago she became the patient of the late Dr. Sidney Yankauer, who was similarly unsuccessful in arresting the growth or recurrence of the polypi. In addition to various and repeated intranasal operations, she had been

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treated by means of electrocautery, diathermy, vaccines, application of various chemicals, powders, ointments and irrigations, but without effect. The blood Wassermann and sensitization tests were negative.

Ten years ago she had radiotherapy, which included implantation of radium seeds and a series of X-ray treatments. For the first time the rapidity of the growth was

Fig. 1A. Family group at the age of 10 years. Note that she is the only mouth-breather in the group. At this time there was already the history of polyposis.

Fig. 1B. At the age of 26 years. Note the rather large nose.

Figs. 1C and 1D. At the age of 50 years. Note the expansion of the nose.

inhibited so that polypectomy was necessary only monthly in order to keep her comfortable.

Nine years ago Dr. Yankauer assigned this patient to me. At that time her nose was broadened, the overlying skin was moderately thickened and somewhat elevated. Intranasally, both nostrils were completely occluded by polypi so that it was impossible to see the interior of the nose. The polypi were smeared with thick, yellow, adherent pus. The posterior nasal examination revealed choanae completely filled

with polypi. Following the removal of numerous polypi, the interior of the nose became visible. Firmer tissue was present on the roof of the nasal chamber, a perforation of the posterior part of the septum was completely traversed by polypi, and the mucous membrane of the floor had an irregularly wrinkled appearance. Her symptoms were again relieved for a short period. She returned as usual for polypectomy because of the recurrences.

Three years ago I suggested a massive polypectomy, which was done at the Mount Sinai Hospital. X-rays were taken

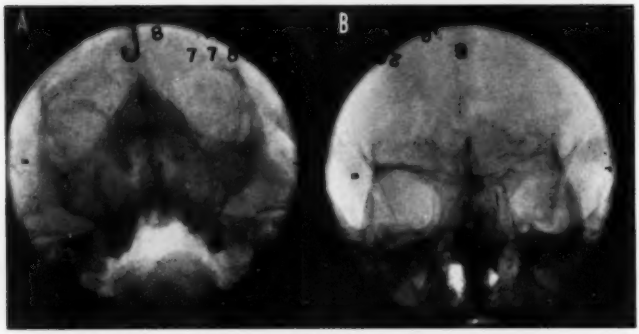

Fig. 2A. Marked clouding of both antra; peculiar streaked mottling of the frontal sinuses is seen. Other views show same condition in the sphenoids. X-rays made in 1934.

Fig. 2B. Attempt made to instill lipiodol shows it to be present in the nostrils only.

of her sinuses, which disclosed a marked clouding and mottling of all the sinuses, suggesting a pansinus polyposis. At the time of operation hundreds of polypi were removed, most of which were very small. A microscopic examination was made and the pathologist reported inflamed mucous polypi. She was rather comfortable for a long interval and with the recurrence of the polypi a similar procedure was carried out at the Beekman Street Hospital the following year. Tissue examined again revealed mucous polypi. Last year the nose again became markedly obstructed and another polypectomy was performed. The tissues were sent to the laboratory for examination. This time the report was carcinoma.

The patient was then referred to Dr. William Harris for deep X-ray therapy. Following radiotherapy the nose became roomier, the secretions diminished. The patient breathed well and was very happy. About six weeks ago she began to experience pain for the first time. The left side of her face was swollen, painful and tender. The swelling began at the zygoma and extended down over the cheek. Nasal examination revealed ulceration and polypoid tissue in the

Fig. 2C. X-rays made in 1937, showing marked clouding of both antra with destruction of the left antrum. There is an irregular clouding of all the sinuses.

left nasal cavity just above the level of the middle turbinate. The left antrum was irrigated and the return was sanguineous. An X-ray was taken, showing a breaking down of the antral wall. The tumor is now expanding and breaking through the floor and anterior wall of the antrum. Another biopsy was recently taken from the nose and also from the antrum through a previous operative opening. This was reported infiltrating squamous-cell carcinoma.

Pathology: The tissue removed at the first biopsy revealed the typical pathologic finding of a nasal polyp. The lining was composed of regular cylindrical epithelium placed upon

a hyaline basement membrane, and frequently consisted of several layers. The cells were uniform and parallel to each other. The nuclei of the cells were oval in shape and contained a moderate amount of chromatin. The stroma was edematous and infiltrated with polymorphonuclear leukocytes, abundant eosinophiles, plasma cells and lymphocytes. Many thick-walled vessels could be seen.

Biopsy of the section which was removed one year later showed a slight change in the epithelium. The importance of these early changes was evaluated after the last tissue

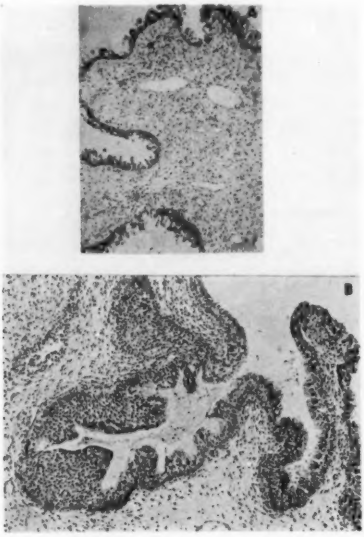

Fig. 3A. Edematous mucous polyp.
Fig. 3B. Showing squamous-cell metaplasia of surfaces.

specimen was examined and showed the malignant metaplasia. This specimen showed the tissue to be lined by several layers of cylindrical epithelium, which occasionally dipped deeply into the underlying stroma. The cells were regularly cylindrical as a rule, but occasional cells which were more polygonal could be seen. These cells resembled squamous epithelial cells. The stroma was unaltered.

The tissue removed at the polypectomy from which the diagnosis of carcinoma was made showed it to be lined by cylindrical epithelium. There were several nests, however, in which the surface epithelium formed broad plaques composed of squamous epithelium. The latter were flattened by contact, but showed intercellular bridges. These cells showed striking nuclear irregularities in regard to size and chromatin content. There were even giant forms present. Sporadic

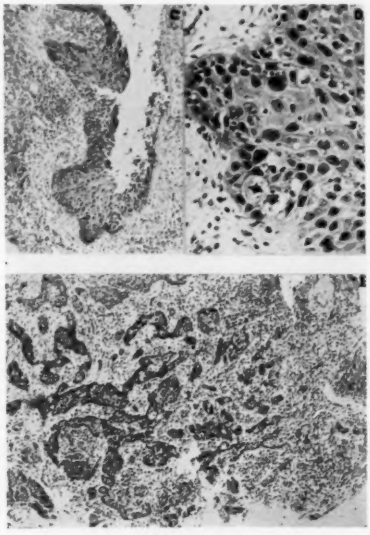

Fig. 3C. Showing carcinomatous changes.
Fig. 3D. Higher magnification of previous picture, showing marked cellular irregularity or atypism with mitosis.
Fig. 3E. Showing definite infiltrating strands of squamous-cell carcinoma.

mitoses were seen. The stroma was unaltered. The last specimen, removed one week ago, was very striking. It consisted of several fragments of tissue, most of which showed infiltration of the stroma with strands of squamous-cell carcinoma. There were, however, fragments which still showed the surface to be lined by columnar epithelium with nests of anaplastic squamous cells. The stroma was densely infiltrated with leukocytes, plasma cells and lymphocytes.

COMMENT.

This is the first recorded case in which polyposis has been observed continuously for over four decades, with final transformation into a malignant phase. The only other case of long standing polyposis with malignant degeneration which I have been able to find in the literature was that of Wagers', reported in 1924. His patient was age 55 years, with a history of nasal obstruction for 30 years, who had been treated only for the past three years. Polypi apparently benign, but from which no microscopic sections were made, were removed on three occasions. Wagers found extensive polypoid involvement, but microscopic examination of the tissue revealed squamous-cell carcinoma.

Other authors have reported carcinomata in association with polypi, where they had been known to exist for a shorter time. Robinson reported transformation into angiofibrosarcoma; Crowe reported a similar change in a second case. Crowe and Baylor reported a case where carcinoma was found in the mucous membrane at the point of attachment of the polypi, but felt that the polypi were incidental to and not a forerunner of the malignant growth.

Malignant degeneration of polypi in the intestinal tract, bladder and uterus is not uncommon. Some consider polypi in these regions precancerous. On the other hand, malignant metaplasia of nasal polypi has been questioned by pathologists as well as rhinologists. Ewing has stressed the absence of neoplastic features of a persistent and progressive polyp. Changes in the appearance and proportion of cells are seldom pronounced and he, therefore, classifies the polyp as the purest example of pseudoneoplasm. The association of polypi with malignant cases has been looked upon by others as incidental or as a result of the primary growth. This present case and that of Wagers' show that polypi may be carcinogenic. The factors involved in the metaplasia are, of course, indefinite, but there are several possibilities which should be enumerated, such as chronic inflammation and irritation from pus, trauma occasioned by frequent operative procedures, earlier application of caustics and the influence of radiotherapy as a stimulant.

Finally, it should be emphasized that, although polypi are considered to be benign, recurrences, especially in cases where

they are persistent and out of proportion to the apparent disease, should call for repeated microscopic examination. Since nasal polypi may undergo malignant changes, microscopic examination will be valuable in diagnosis so that early treatment may be instituted before the tumor begins to spread.

SUMMARY.

A case is reported in which polypi of the nose under medical observation for 42 years, uncontrollable and persistently recurring, became carcinomatous. Malignant metaplasia from very slight epithelial changes to infiltrating squamous-cell carcinoma is demonstrated by a series of microscopic sections made at successive biopsies.

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THE FISHOF NEEDLE.*

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In 1935, when I started the injection treatment of vasomotor rhinitis with sodium morrhuate, after trying the usual needles, I found it necessary to devise a special one for this purpose. It is (Fig. 1) a double curved needle, so constructed that it conforms to the anatomical structure of the nasal turbinates. A line drawn from the point of the needle to its hub forms an equilateral parallelogram, so that when another line is drawn to connect the upper and lower angles, it runs through the centre of the longitudinal axis of the syringe. The curve near the hub has an angle of 20°, and the second bend near the point has the same angle. This construction has two advantages: *1.* Field of vision is unobstructed. *2.* Prevents bending or breaking of the needle, because the force applied in injecting is carried in a straight line through the centre of the hub of needle and syringe.

The bending and breaking of the needle is further protected by making it of hyperchrome steel, which is not only rust-resisting but also has unusual resistance to breakage. This hyperchrome extends through the entire length of the extension, from the hub to the tip. In this way air sockets and the collection of foreign matter are prevented.

The Fishof Needle is made in 20 and 21 gauge. By use of this needle (see Figs. 2, 3, 4 and 5) one can readily inject the medial or lateral surface, the superior or inferior border of any turbinate by merely rotating it to an arc of 90°. The convexity of the lower bend faces the surface that one wishes to inject. The posterior tip of the turbinate can be reached with ease, either for injection or aspiration purposes.

Septums that are markedly deviated or spurred can be readily injected with this instrument. The double bayonet-like shape of the needle leaves a clear field of vision for

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*This needle was developed in the course of work done in the E., N. and T. Department of the Hospital for Joint Diseases and Service of Dr. Alfred Braun, and was presented to the medical profession on May 4, 1937, at the Clinical Society of the Hospital for Joint Diseases.

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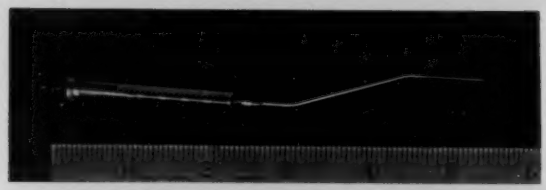

Fig. 1.

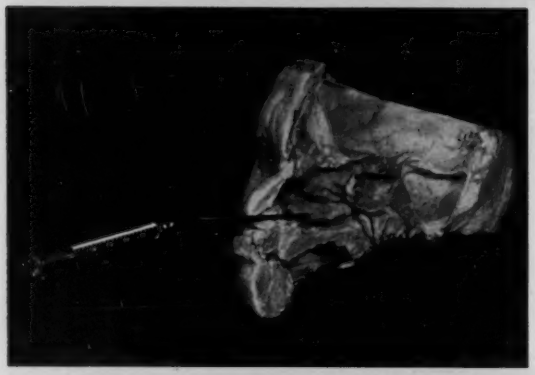

Fig. 2. Needle in medial surface of right inferior turbinate.

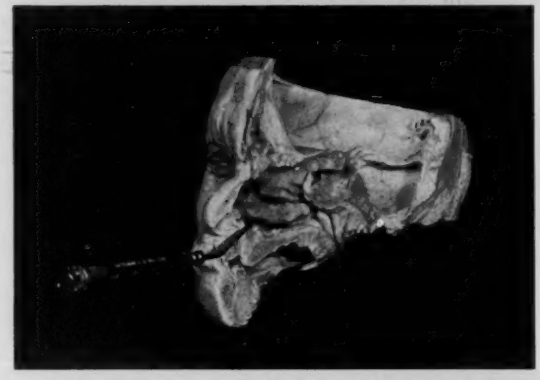

Fig. 3. Needle in lateral surface of right inferior turbinate.

injecting the ethmoid and sphenoid regions. It is to be noted that gauge 20 is preferred when fluid is to be injected close to the periosteum; gauge 21 is selected when the tis-

sues are hyperemic or when injecting polypoid structures or hyperplastic turbinates.

Briefly stated, this is a double curved or double bayonet-like shaped needle, so constructed that it enables one to inject

Fig. 4. Needle in superior border of right inferior turbinate.

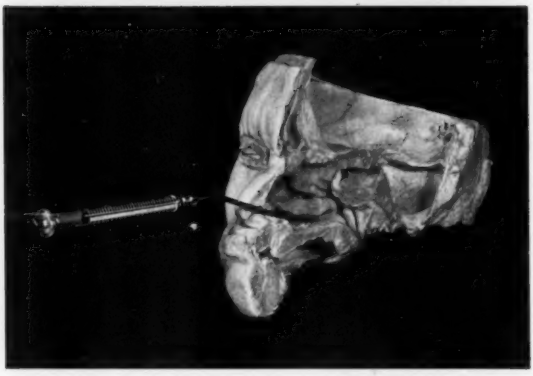

Fig. 5. Needle in inferior border of right inferior turbinate.

with ease and safety the turbinates, ethmoid and sphenoid regions, and markedly deviated and badly spurred septa; in other words, it is a universal needle well adapted for injecting structures in the intranasal vault.

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